A Dissertation on

# '' "THE ROLE OF LOW MOLECULAR WEIGHT HEPARIN IN TREATMENT OF ACUTE PANCREATITIS''

Dissertation Submitted to

**THE TAMIL NADU Dr.M.G.R. MEDICAL UNIVERSITY**

**CHENNAI- 600032**

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**M.S. GENERAL SURGERY**

**(BRANCH 1)**

**COIMBATORE MEDICAL COLLEGE,**

**COIMBATORE**

**MAY 2018**

# CERTIFICATE

This is to certify that this dissertation titled **"THE ROLE OF LOW MOLECULAR WEIGHT HEPARIN IN TREATMENT OF ACUTE PANCREATITIS "** is the bonafide work of Dr.ARAVIND. M Post Graduate student in M.S General Surgery, Coimbatore Medical College and Hospital, Coimbatore. This study was undertaken in the Department of General Surgery, Coimbatore Medical College and Hospital, Coimbatore during the period June 2016 to August 2017 in the partial fulfillment of the requirement of the "The Tamil Nadu Dr. M.G.R. Medical University" for the award of M.S. Degree in General Surgery. This dissertation has not been submitted in part or fully to any other University or Board. It gives me great pleasure to forward this dissertation.

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# DECLARATION

The dissertation titled **"THE ROLE OF LOW MOLECULAR WEIGHT HEPARIN IN TREATMENT OF ACUTE PANCREATITIS"** is being submitted by me to "The Tamil Nadu Dr. M.G.R. Medical University" in partial fulfillment of the regulation for the for award of of M.S. Degree in General Surgery (Branch – 1). This work has been carried out in the Department of General Surgery, Coimbatore Medical College and Hospital, Coimbatore under the guidance of **Dr.S.Balasubramanian M.S,** Professor of General Surgery, Coimbatore Medical College and Hospital, Coimbatore.

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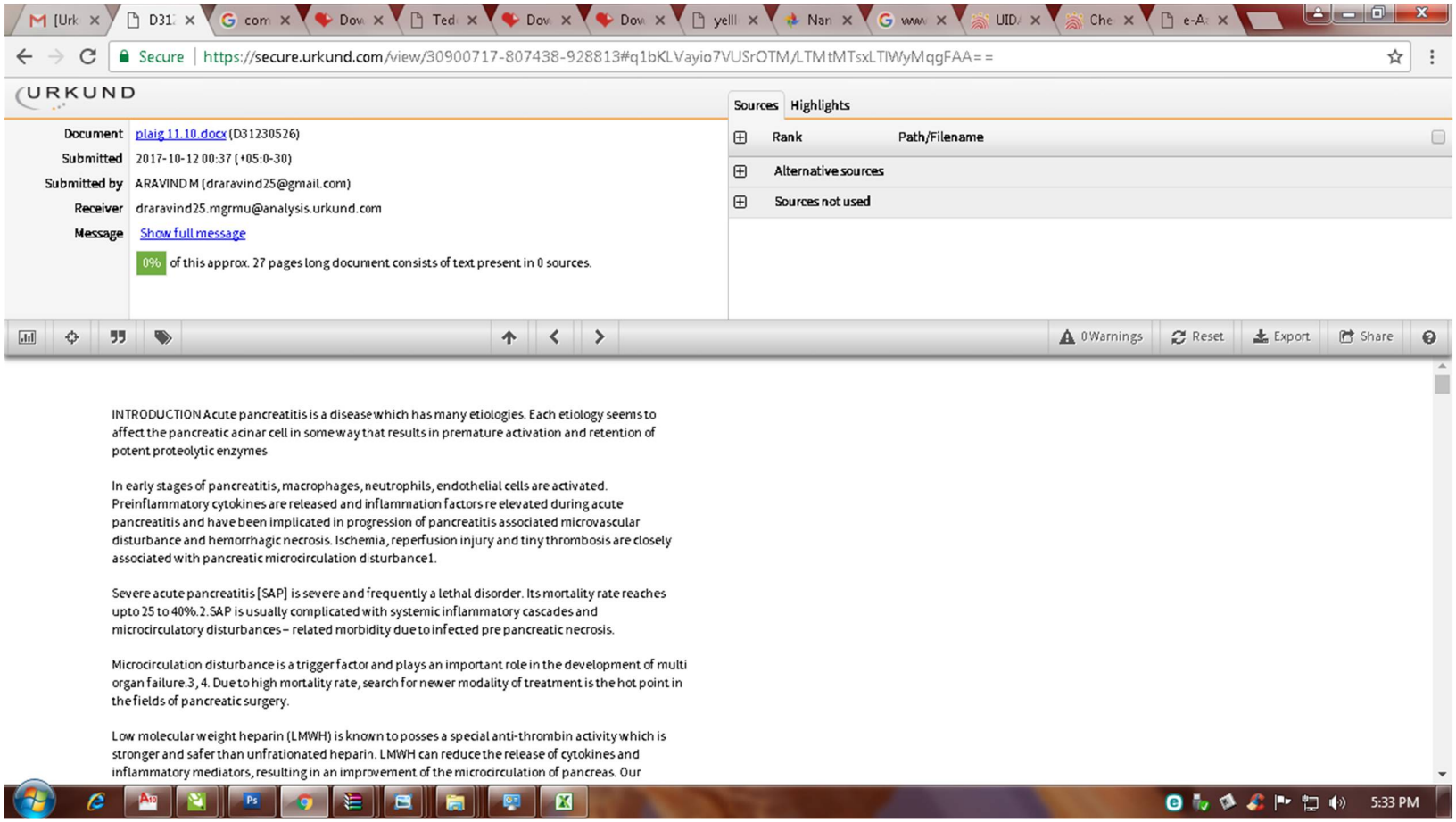
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INTRODUCTION Acute pancreatitis is a disease which has many etiologies. Each etiology seems to affect the pancreatic acinar cell in some way that results in premature activation and retention of potent proteolytic enzymes
In early stages of pancreatitis, macrophages, neutrophils, endothelial cells are activated. Preinflammatory cytokines are released and inflammation factors re elevated during acute pancreatitis and have been implicated in progression of pancreatitis associated microvascular disturbance and hemorrhagic necrosis. Ischemia, reperfusion injury and tiny thrombosis are closely associated with pancreatic microcirculation disturbance1.
Severe acute pancreatitis [SAP] is severe and frequently a lethal disorder. Its mortality rate reaches upto 25 to 40%.2.SAP is usually complicated with systemic inflammatory cascades and microcirculatory disturbances – related morbidity due to infected pre pancreatic necrosis.
Microcirculation disturbance is a trigger factor and plays an important role in the development of multi organ failure.3, 4. Due to high mortality rate, search for newer modality of treatment is the hot point in the fields of pancreatic surgery.
Low molecular weight heparin (LMWH) is known to posses a special anti-thrombin activity which is stronger and safer than unfrationated heparin. LMWH can reduce the release of cytokines and inflammatory mediators, resulting in an improvement of the microcirculation of pancreas. Our
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## CERTIFICATE – II

This is to certify that this dissertation work titled **"THE ROLE OF LOW MOLECULAR WEIGHT HEPARIN IN TREATMENT OF ACUTE PANCREATITIS"** of the candidate **Dr.ARAVIND. M** with registration Number **221511302** for the award of **M.S in the branch of General Surgery**,I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains 110 pages from introduction to conclusion and the result shows **0% (Zero)** percentage of plagiarism in the dissertation.

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# ABBREVIATIONS

| | | |
|---|---|---|
| APACHE | : | Acute Physiology And Chronic Health Evaluation |
| BISAP | : | Blood Urea Impaired Mental Status SIRS Age Pleural Effusion |
| CARS | : | Counter Anti Inflammatory Response Syndrome |
| CBD | : | Common Blie Duct |
| CECT | : | Contrast Enhanced Computed Tomography |
| CKD | : | Chronic Kidney Disease |
| CTSI | : | Computed Tomograph Severity Index |
| ERCP | : | Endoscopic Retrograde Cholangio Pancreatography |
| MCTSI | : | Modified Computed Tomograph Severity Index |
| MODS | : | Multi Organ Dysfunction |
| MRI | : | Magnetic Resonance Imaging |
| SAP | : | Severe Acute Pancreatitis |
| SIRS | : | Severe Inflammatory Response Syndrome |

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# INTRODUCTION

Acute pancreatitis is a disease which has many etiologies. Each etiology seems to affect the pancreatic acinar cell in some way that results in premature activation and retention of potent proteolytic enzymes

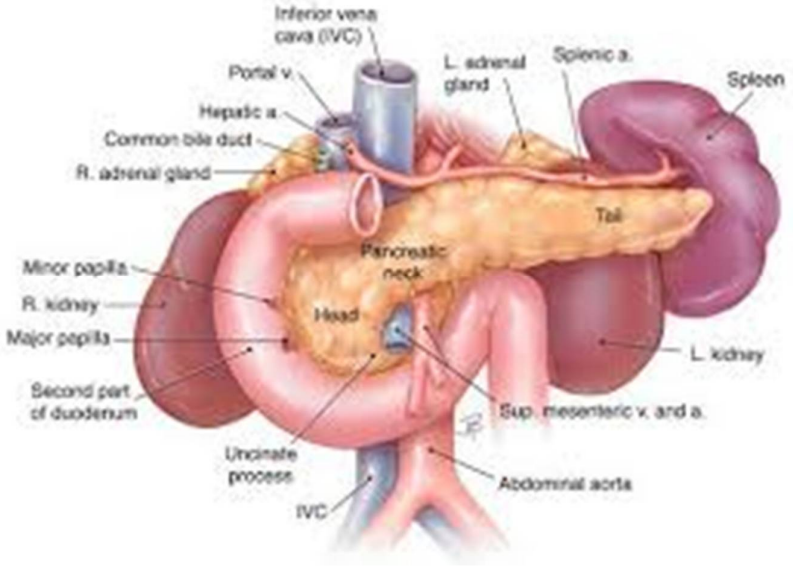
In early stages of pancreatitis, macrophages, neutrophils, endothelial cells are activated. Preinflammatory cytokines are released and inflammation factors re elevated during acute pancreatitis and have been implicated in progression of pancreatitis associated microvascular disturbance and hemorrhagic necrosis. Ischemia, reperfusion injury and tiny thrombosis are closely associated with pancreatic microcirculation disturbance[1].

Severe acute pancreatitis [SAP] is severe and frequently a lethal disorder. Its mortality rate reaches upto 25 to 40%.[2].SAP is usually complicated with systemic inflammatory cascades and microcirculatory disturbances – related morbidity due to infected pre pancreatic necrosis.

Microcirculation disturbance is a trigger factor and plays an important role in the development of multi organ failure.[3, 4]. Due to high mortality rate, search for newer modality of treatment is the hot point in the fields of pancreatic surgery.

Low molecular weight heparin (LMWH) is known to posses a special anti-thrombin activity which is stronger and safer than unfrationated heparin. LMWH can reduce the release of cytokines and inflammatory mediators, resulting in an improvement of the microcirculation of pancreas.

Our experimental study provides evidence that LMWH can block the initiation of an inflammatory storm, leading to improvement of microcirculation system; and has anti-thrombus effect to reduce the formation of microthrombosis in pancreas. These findings demonstrate the important therapeutic effect of LMWH in the treatment of acute pancreatitis



## AIM OF THE STUDY

Effect of LMWH in improvement of microcirculation in acute pancreatitis.

## OBJECTIVES OF THE STUDY

To study the effect of low molecular weight heparin (LMWH) in the treatment of acute pancreatitis.

# REVIEW OF LITERATURE

## A. Role Of Low Molecular Weight Heparin In Treatment Of Acute Pancreatitis

- A multiple centre prospective clinical study done to assess the effect of Low Molecular Weight Heparin in the treatment of severe acute pancreatitis published in 2009[5] used a total of 265 SAP patients who were randomly divided into two groups: firstly, the conventional treatment group (C group, n = 130); and secondly the conventional treatment plus the LMWH treatment group (LT group, n = 135). The clinical parameters, laboratory parameters and computed tomography(CT) score of pancreatic necrosis (CTSPN) in the two groups were compared.On admission, all the clinical parameters, laboratory parameters and CTSPN in the two groups were not significantly different ($p > 0.05$). However, after treatment, in LT group, the clinical presentation improvement rate and laboratory parameters improvement were significantly higher than those in C group ($p < 0.05–0.01$), and the acute physiology and chronic health evaluation (APACHE) II score, complication rate, mortality and mean hospital stay in LT group were obviously lower than those in C group ($p < 0.05–0.01$). The CT score in LT group was much lower than that in C group ($p < 0.05$). Two weeks after treatment FBI decreased

obviously in C group, but not in LT group, and no hemorrhagic complications occurred. So, it was concluded that LMWH can enhance the effect of conventional treatment for SAP, and can markedly decrease the mortality of SAP. LMWH is a simple, safe, economic and effective method for treatment of SAP[(5)].

- A study to explore the clinical effects of low-molecular-weight heparin (lmwh) combined with ulinastatin (uti) in children with acute pancreatitis[6].In total, 560 patients with severe acute pancreatitis treated at binzhou people's hospital, shandong, china, from April 2012 to June 2014 were enrolled in this study. They were divided into control (280 patients, ulinastatin + conventional treatment) and observational groups (280 patients, lmwh + ulinastatin + conventional treatment). The treatment lasted for 2 weeks. Clinical parameters, laboratory test indices, acute physiology and chronic health evaluation (apache ii) score, and computed tomography score of pancreatic necrosis (ctspn) were assessed in both groups. On admission, no significant differences were noted in clinical features, laboratory parameters, apache ii scores, or ctspn between the two groups (all $p > 0.05$). After 2 weeks of treatment, serum amylase, urine amylase, prothrombin time, fibrinogen, partial thromboplastin time, and platelet count in the study group were 913 ± 281 u/l, 1893 ±

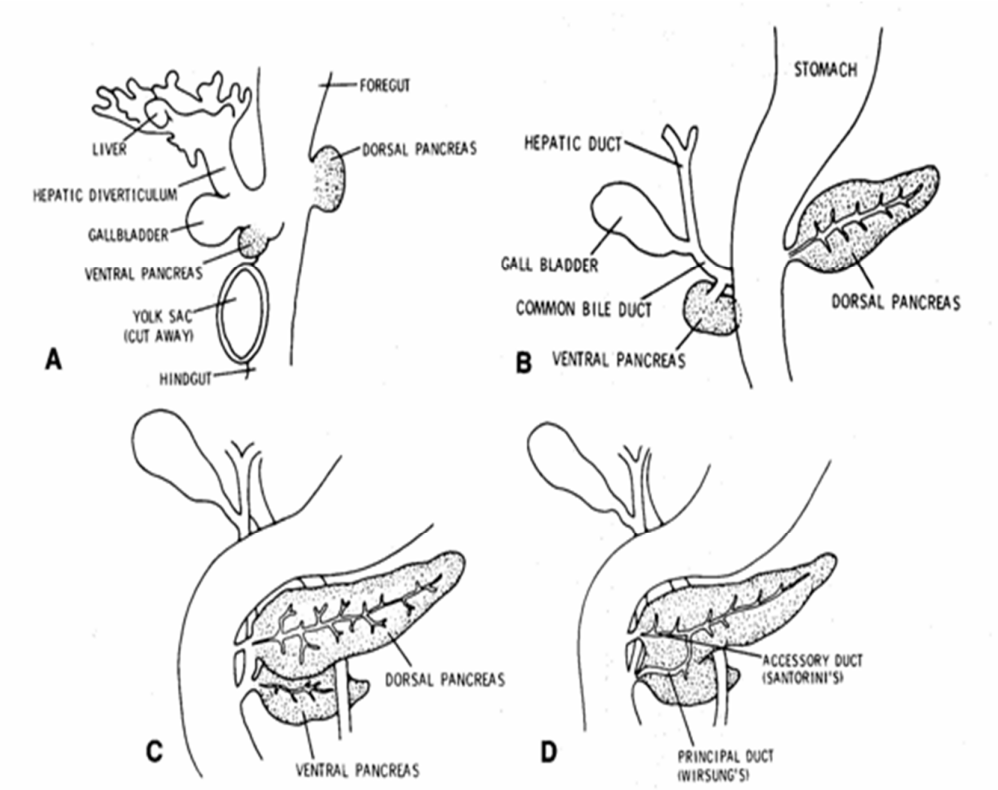
295 u/l, 16 ± 1.60 s, 3 ± 0.60 g/l, 39.80 ± 5.60 s, and 294 ± 49 × $10^9$/l, respectively, all of which were similar or superior to those in the control group (1738 ± 346 u/l, 2453 ± 473 u/l, 15 ± 1.50 s, 2.50 ± 0.50, 39.80 ± 5.90, and 192 ± 37 × $10^9$/l)). Apache ii scores and ctspn after 2 weeks of treatment in the observation group were 8.50 ± 1.80 and 2.10 ± 1, respectively, which were superior to those in the control group (9.60 ± 2.40 and 4.30 ± 2.60, respectively; $p < 0.05$). Moreover, the incidence of complications, mortality rate, and average duration of the hospital stay in the observation group were lower than those in the control group ($p > 0.05$). The cure rate in the observation group was higher than that in the control group. So, it was concluded lmwh combined with uti enhances the efficacy of conventional treatment and reduces mortality. Thus, it is a potentially effective treatment strategy for severe acute pancreatitis in children[6].

- The current study explored the effects of intensive insulin therapy (IIT) combined with low molecular weight heparin (LMWH) anticoagulant therapy on severe acute pancreatitis (SAP). A total of 134 patients with SAP that received treatment between June 2008 and June 2012 were divided randomly into groups A (control; n=33), B (IIT; n=33), C (LMWH; n=34) and D (IIT + LMWH; n=34). Group A were treated routinely. Group B received continuous pumped insulin,

as well as the routine treatment, to maintain the blood sugar level between 4.4 and 6.1 mmol/l. Group C received a subcutaneous injection of LMWH every 12 h in addition to the routine treatment. Group D received IIT + LMWH and the routine treatment. The white blood cell count, hemodiastase, serum albumin, arterial partial pressure of oxygen and prothrombin time were recorded prior to treatment and 1, 3, 5, 7 and 14 days after the initiation of treatment. The intestinal function recovery time, incidence rate of multiple organ failure (MOF), length of hospitalization and fatality rates were observed. IIT + LMWH noticeably increased the white blood cell count, hemodiastase level, serum albumin level and the arterial partial pressure of oxygen in the patients with SAP ($P<0.05$). It markedly shortened the intestinal recovery time and the length of stay and reduced the incidence rate of MOF, the surgery rate and the fatality rate ($P<0.05$). It did not aggravate the hemorrhagic tendency of SAP ($P>0.05$). IIT + LMWH had a noticeably improved clinical curative effect on SAP compared with that of the other treatment[7]

## Embryology

- The pancreas develops as 2 buds (outpouchings) of endoderm from the primitive duodenum at the junction of the foregut and the midgut. A small ventral bud (pouch) forms the lower (inferior) part of the head and the uncinate process of pancreas, whereas a large dorsal bud (pouch) forms the upper (superior) part of the head as well as the body and tail of the pancreas. The ventral bud rotates behind the duodenum dorsally from right to left and fuses with the dorsal bud, and the duct of the distal part (body and tail) of the dorsal bud unites with the duct of the ventral bud to form the main pancreatic duct (of Wirsung). Because the common bile duct (CBD) also arises from the ventral bud, it forms a common channel with the main pancreatic duct. The remaining proximal part (head) of the duct of the dorsal bud remains as the accessory pancreatic duct (of Santorini).

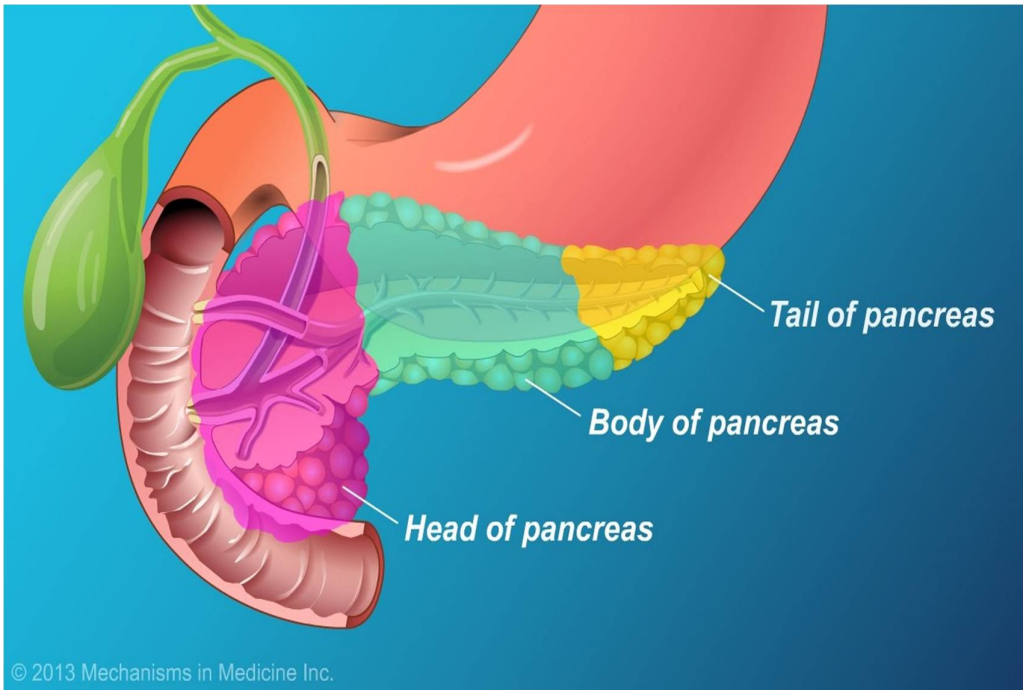

FOREGUT
LIVER
DORSAL PANCREAS
HEPATIC DIVERTICULUM
GALLBLADDER
VENTRAL PANCREAS
YOLK SAC
(CUT AWAY)
A
HINDGUT
STOMACH
HEPATIC DUCT
GALL BLADDER
COMMON BILE DUCT
B
VENTRAL PANCREAS
DORSAL PANCREAS
DORSAL PANCREAS
C
VENTRAL PANCREAS
ACCESSORY DUCT
(SANTORINI'S)
D
PRINCIPAL DUCT
(WIRSUNG'S)

## Gross Anatomy

The pancreas is prismoid in shape and appears triangular in cut section with superior, inferior, and anterior borders as well as anterosuperior, anteroinferior, and posterior surfaces.

The head of the pancreas lies in the duodenal C loop in front of the inferior vena cava (IVC) and the left renal vein (see the following images). The uncinate process is an extension of the lower (inferior)

half of the head toward the left; it is of varying size and is wedged between the superior mesenteric vessels (vein on right, and artery on left) in front and the aorta behind it.



The lower (terminal) part of the CBD runs behind (or sometimes through) the upper half of the head of pancreas before it joins the main pancreatic duct (MPD) of Wirsung to form a common channel (ampulla).

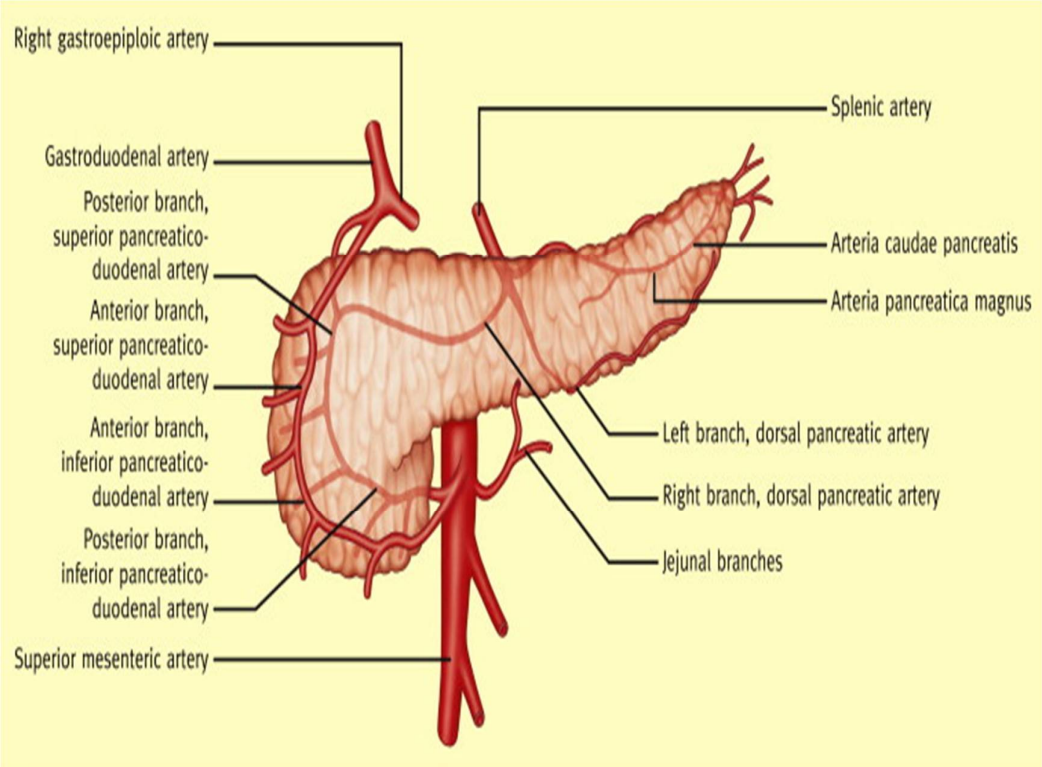
The neck of the pancreas lies in front of the superior mesenteric vein, splenic vein and portal vein junction. The body and tail of the pancreas run obliquely upward to the left in front of the aorta and left kidney. The pancreatic neck is the arbitrary junction between the head and body of the pancreas. Portal vein lies behind the neck of the pancreas. The narrow tip of the tail of the pancreas reaches the splenic hilum in the splenorenal (lienorenal) ligament.

The pancreatic head constitutes about 50% and the body and tail the remaining 50% of the pancreatic parenchymal mass.

The transverse mesocolon (with the middle colic vessels in it) is attached to the anterior surface of the lower (inferior) part of body and tail—most of the gland is thus located in the supracolic compartment. The body and tail of the pancreas lie in the lesser sac (omental bursa) behind the stomach

**Blood supply**

Pancreas derives a rich blood supply from both celiac axis and superior mesenteric artery; that is why when angiography is done for bleeding as a complication of acute pancreatitis, chronic pancreatitis or pancreatoduodenectomy both celiac axis and superior mesenteric artery should be evaluated.




The celiac trunk (axis) comes off from the anterior surface of the aorta at the level of T12–L1. It has a short length of about 1 cm and trifurcates into the common hepatic artery (CHA), splenic artery, and left gastric artery (LGA). The CHA runs toward the right on the superior border of

the proximal body of the pancreas, and the splenic artery runs toward the left on the superior border of the distal body and tail of the pancreas. [1, 2, 3, 4, 5]

The superior mesenteric artery (SMA) comes off from the anterior surface of the aorta just below the origin of the celiac trunk at the level of L1 behind the neck of the pancreas. Then, it descends down in front of the uncinate process and the 3rd (horizontal) part of the duodenum to enter the small bowel mesentery.

The gastroduodenal artery (GDA), a branch of the CHA, runs down behind the first part of the duodenum in front of the neck of the pancreas and divides into the right gastro-omental (gastroepiploic) artery (RGEA) and superior pancreaticoduodenal artery (SPDA), which further bifurcates into anterior and posterior branches. The inferior pancreaticoduodenal artery (IPDA) arises from the SMA and also bifurcates into anterior and posterior branches.

The anterior and posterior branches of the SPDA and IPDA join each other and form anterior and posterior pancreaticoduodenal arcades in the anterior and posterior pancreaticoduodenal grooves supplying small branches to the pancreatic head and uncinate process of the pancreas as well as the 1st, 2nd, and 3rd parts of the duodenum (vasa recta duodeni). Multiple pancreatic branches (including a great pancreatic artery or

arteria magna pancreatica) of the splenic artery supply the pancreatic body and tail. Multiple, small pancreatic branches of a dorsal pancreatic artery from the splenic artery and an inferior pancreatic artery from the superior mesenteric artery supply the body and tail of pancreas.

The arterial supply of the pancreas forms an important collateral circulation between the celiac axis and superior mesenteric artery.

Veins accompany the SPDA and IPDA. Superior pancreaticoduodenal veins (SPDVs) drain into the portal vein and inferior pancreaticoduodenal veins (IPDVs) drain into the superior mesenteric vein (SMV). A few small, fragile uncinate veins drain directly into the SMV. Some veins from the head of the pancreas drain into the gastrocolic trunk. Numerous small, fragile veins drain directly from the pancreatic body and tail into the splenic vein.

The SMV lies to the right of the SMA in front of the uncinate process and the 3rd part of the duodenum. The splenic vein arises in the splenic hilum behind the tail of the pancreas and runs from left to right on the posterior surface of the pancreatic body. Union of the horizontal splenic vein and the vertical SMV forms the portal vein behind the neck of the pancreas.

The inferior mesenteric vein (IMV) joins the splenic vein (or the junction of the splenic vein and SMV, or even SMV). The portal vein receives the

SPDVs, right gastro-omental (gastroepiploic vein, left gastric vein (LGV), and right gastric vein (RGV); then, it runs up (superiorly) behind the first part of the duodenum in the hepatoduodenal ligament behind (posterior to) the common bile duct on the right and proper hepatic artery on the left.

The portal venous system (splenic vein, SMV, and portal vein) has no valves.

## Lymphatic drainage

The head of the pancreas drains into pancreaticoduodenal lymph nodes and lymph nodes in the hepatoduodenal ligament, as well as prepyloric and postpyloric lymph nodes. The pancreatic body and tail drain into mesocolic lymph nodes (around the middle colic artery) and lymph nodes along the hepatic and splenic arteries. Final drainage occurs into celiac, superior mesenteric, and para-aortic and aortocaval lymph nodes.

### Nerve supply

The pancreas receives parasympathetic nerve fibers from the posterior vagal trunk via its celiac branch. Sympathetic supply comes from T6-T10 via the thoracic splanchnic nerves and the celiac plexus.

## Natural and Pathophysiologic Variants

### Natural variants

The main pancreatic duct and common bile duct may not unite to form a common channel and open separately at the major duodenal papilla. In addition, an aberrant (normal vessel is not present) right hepatic artery (RHA) may arise from the superior mesenteric artery (SMA) and accessory RHA (in addition to the normal one from common hepatic artery [CHA]) from the SMA.

### Pathophysiologic variants

An annular pancreas is caused by failure of rotation of the ventral bud of the pancreas. A ring of pancreas is present around and obstructs the second part (C loop) of the duodenum. Neonates with this pancreatic variant present with vomiting; abdominal x-rays show a double-bubble (gastric and duodenal) appearance. Treatment includes dudodeno-jejunostomy and not division of the pancreatic ring because it may result in pancreatic juice leak and fistula.

Pancreas divisum is due to failure of the main and accessory pancreatic ducts to fuse. In addition to the upper (superior) half of the head of pancreas (which it normally also drains), the accessory pancreatic duct (of Santorini) also drains the body and tail of pancreas. This drainage

may not be adequate (because of the smaller size of the accessory duct) and may cause functional obstruction, resulting in recurrent attacks of acute pancreatitis. The main pancreatic duct (of Wirsung) drains only the lower (inferior) half of the head and uncinate process and does not communicate with the accessory duct.

A long (> 15 mm common channel of pancreatic duct and common bile duct is described as anomalous pancreatio biliary ductal junction/ union (APBDJ/ APBDU) - it is associated with choledochal cyst and carries a higher risk of biliary malignancy.

Accessory pancreatic tissue may be present in the stomach, small intestine, Meckel diverticulum, omentum, and hilum of spleen as soft yellow nodules/lobules.

**Physiology**

Pancreas is one of the organs in the body that has both exocrine and endocrinal functions. The function of the pancreas is to make digestive enzymes which digest food materials in the small intestines. In addition the pancreas also makes insulin which controls the blood glucose levels.

**Exocrine Function**

The adult pancreas produces 10 -20g of pancreatic enzymes in an active form.It secretes a colorless odorless alkaline (pH 7.6-9) juice with

enzymes such as – amylase, lipase, proteases and trypsinogen which help digest the fat, protein as well as carbohydrates from the food that we eat. The alkaline juice helps to neutralize the acid secretions of the stomach. It secretes about 1.5 liters of these juices in a day. The stimulants for enzymes secretion are secretin, cholecystokinin and acetylcholine.

The enzymes are conveyed to the upper part of the small intestine called duodenum via a tube called the pancreatic duct.

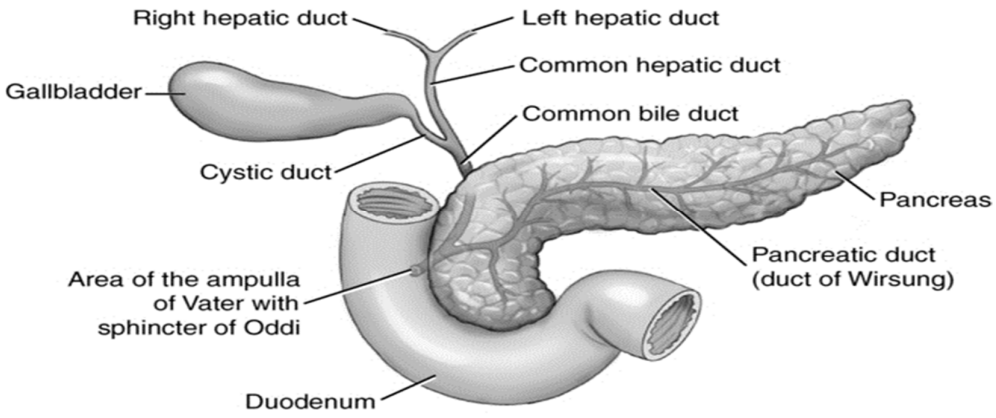
**Endocrine Function**

It also secretes two important hormones namely – Insulin and Glucagon which are essential for regulation of glucose in the blood. Insulin is synthesizes by the B cells. Proinsulin is transported from the endoplasmic reticulum to the Golgi complex where it is packaged into granules and cleaved into insulin and residual connective peptide (C peptide). Glycogen is secreted by the A cells of the islet. It elevates blood glucose levels through the stimulation of glycogenolysis and gluconeogenesis. Its major stimulants are aminoacids and cholinergic fibers. Somatostatin is secreted by the D cells of the islet. It inhibits the release of growth hormone and almost all peptide hormones. It also inhibits gastric, pancreatic and biliary secretion.

## B. Definition of acute pancreatitis

The International Symposium on Acute Pancreatitis (Atlanta, September 1992) defined Acute Pancreatitis as "An acute inflammatory process of the pancreas, with variable involvement of other regional tissues or remote organ systems" [8]

Acute Pancreatitis is an inflammatory disease of the pancreas that is associated with little or no fibrosis of the gland and by definition, it is reversible. It is distinguished from chronic pancreatitis by the absence of continuing inflammation, irreversible structural changes and permanent impairment of exocrine and endocrine pancreatic function. Acute pancreatitis can be initiated by several factors, including gallstones, alcohol, trauma and infections and in some cases, it is hereditary. Very often patients with acute pancreatitis develop additional complications such as sepsis, shock, respiratory and renal failure, resulting in considerable mortality and morbidity.




## C. Classification of Acute Pancreatitis

A commonly used classification system (the Atlanta classification) [8] divides AP into two broad categories. The classification was developed following 3 days of group meetings and open discussions, with unanimous consensus on a series of definitions for a clinically based classification system for acute pancreatitis by a diverse group of 40 international authorities from six medical disciplines and 15 countries. The proposed classification system was to be of value to practicing clinicians in the care of individual patients and to academicians seeking to compare inter-institutional data.

**The Atlanta Classification:**

**(1) Mild :** (edematous and interstitial) acute pancreatitis

**(2) Severe:** (usually synonymous with necrotizing) acute pancreatitis

The criteria for severe AP included any of the following:

1) A Ranson's score of 3 or more

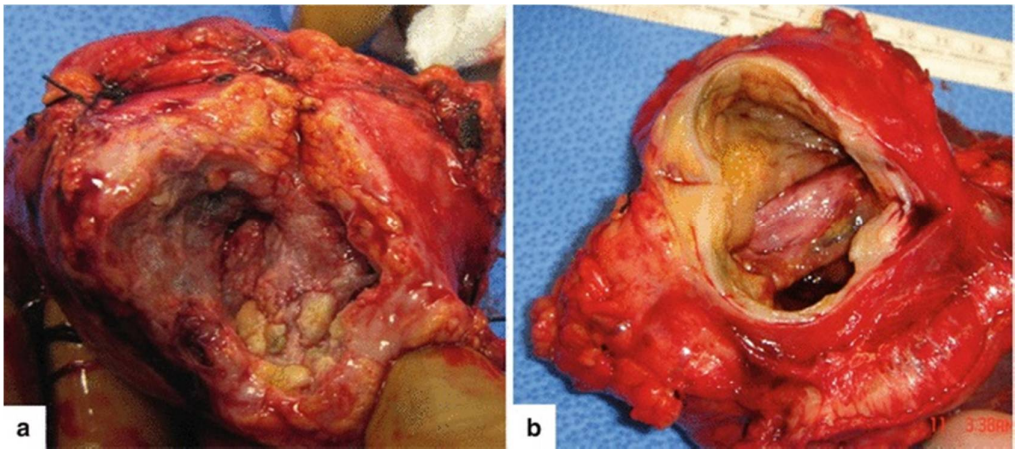
2) An APACHE II score of 8 or more within the first 48 hours
3) Organ failure (respiratory, circulatory, renal, and/or GI bleeding)
4) Local complications (pancreatic necrosis, abscess or pseudocyst)

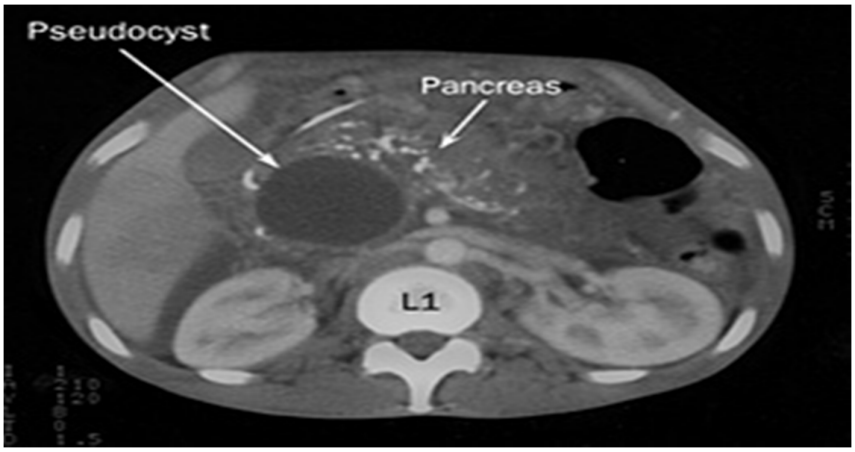
Most attacks of AP are mild with recovery occurring within five to seven days. Death is unusual (less than 3 percent) in such patients. [9]

Approximately 15 to 25 percent of all cases are severe. Severe necrotizing pancreatitis is associated with a high rate of complications (local and systemic) and mortality (approximately 17%)

**Severe Acute Pancreatitis as defined by Atlanta Symposium:**

- Early prognostic signs
- Ranson's score $\geq$3
- APACHE II score $\geq$8
- Organ failure and/or Local complications
- Necrosis
- Abscess
- Pseudocyst




However some patients with local complications in the absence of organ failure are seen to have low mortality rates, like patients with mild acute pancreatitis, but have prolonged hospitalizations, like patients with severe acute pancreatitis in a new subgroup called "**moderately severe acute pancreatitis"**[(10)]

The Atlanta classification has several deficiencies[(11)] :

- It fails to recognize multiple and persistent (≥ 48 hours) organ failure, which is more predictive of severity than transient organ failure.
- The nomenclature developed to describe local complications cannot be used to describe findings such as walled off pancreatic necrosis and does not allow for the differentiation of infected from sterile necrosis
- It combines predicting systems (early) and local complications (late) to define severe acute pancreatitis.

**Revised Atlanta Classification:**

The Revised Atlanta Classification was initiated as an international, web-based process that began in a clinical symposium in 2007 at the Digestive Diseases Week [12].

Revision of the Atlanta classification was made with an intent to address areas of confusion in the original Atlanta Classification, incorporate modern concepts of the disease, improve clinical assessment of severity, enable standardized data reporting, assist objective evaluation of new treatments, and facilitate communication among treating physicians and different institutions.

**Components**

The Revised Atlanta Classification dealt primarily with two broad areas, namely,

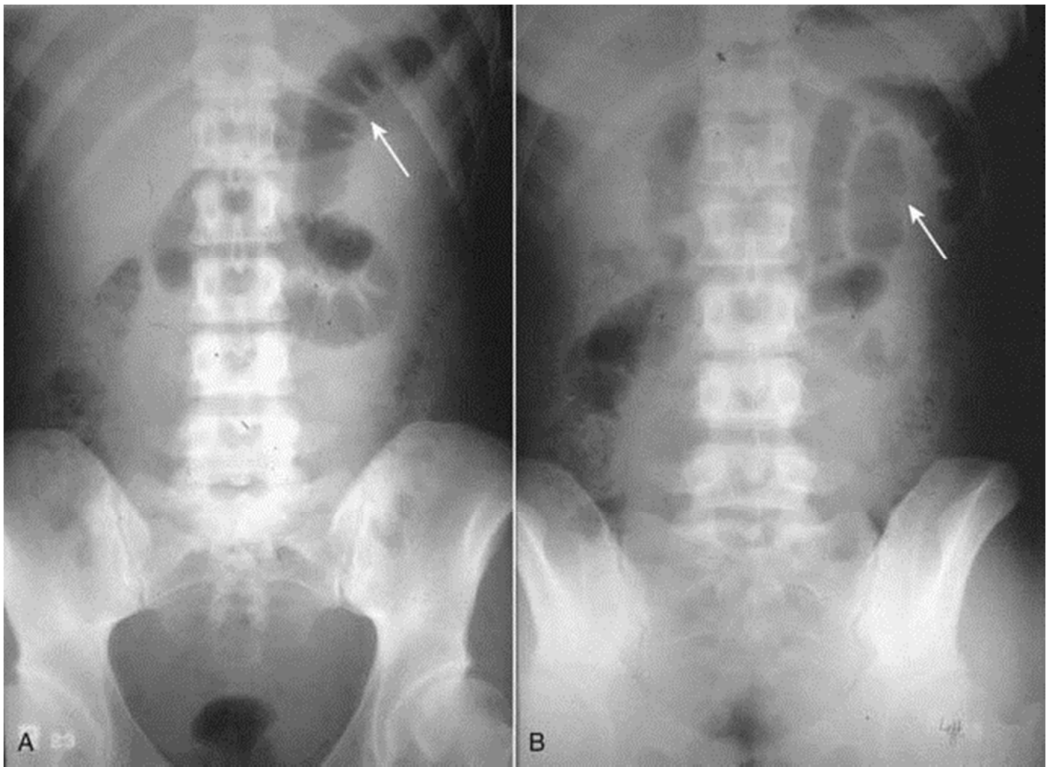
a) discrete definitions of organ failure and local complications (including necrosis) and
b) classification of severity of the disease.

The revised classification categorizes AP into interstitial edematous (IEP) and necrotizing pancreatitis based on contrast enhanced computed tomography (CECT) imaging. IEP constitutes 80-90% of AP, in which the pancreas appears relatively homogenously enhanced on CECT with or without mild peripancreatic stranding or peripancreatic fluid collection. Necrotizing pancreatitis on the other hand is characterized by lack of enhancement of the pancreas and/or (peri) pancreatic tissues on CECT. Both the pancreatic parenchyma and peripancreatic tissues together are involved more frequently than involvement of either alone. Recognition of the degree of necrosis (pancreatic alone, peripancreatic alone, or both) is important since the prognosis varies.

According to the Revised Atlanta Classification, complications of AP can be: organ failure, local and systemic complications.

Organ failure may be transient (resolves within 48 h of onset) or persistent (persists for 48 h and more). Local complications include fluid collections, gastric outlet dysfunction, splenic and portal vein thrombosis, and colonic necrosis. Four discrete types of collections have been described, namely, acute peripancreatic fluid collection (APFC), pancreatic pseudocyst (PP), acute necrotic collection (ANC) and walled off necrosis (WON).

**Sentinel Loop Sign**



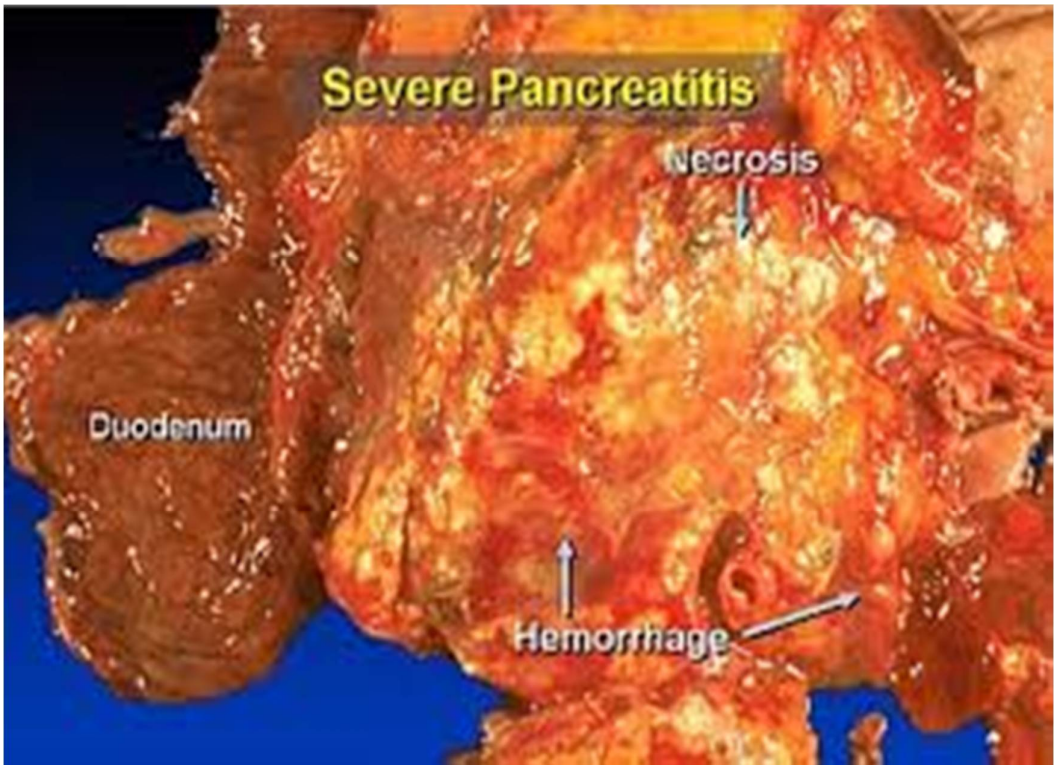
## Colon Cutoff Sign

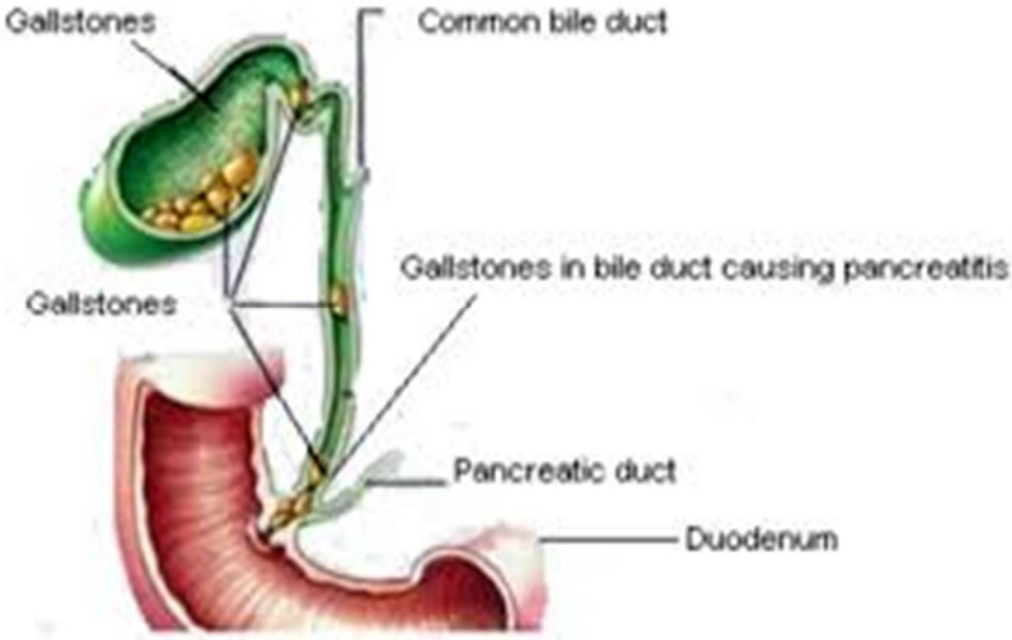
**Table 2: Definition and characteristics of local collections in acute pancreatitis according to the Revised Atlanta Classification**

| Terminology | Definitions and characteristics |
|---|---|
| APFC (acute peripancreatic fluid collection) | • Associated with interstitial edematous pancreatitis<br>• Appear as peripancreatic fluid seen within the first 4 weeks after disease onset.<br>• Does not have a definable wall.<br>• Confined to normal peripancreatic fascial planes.<br>• Does not have intrapancreatic extensions. |
| Pancreatic pseudocyst | • An encapsulated collection of fluid with a well-defined wall.<br>• Usually located outside the pancreas.<br>• Usually requires more than 4 weeks after onset to mature.<br>• Does not contain non-liquid component. |
| ANC (acute necrotic collection) | • Contains variable amounts of both fluid and necrosis.<br>• Associated with necrotizing pancreatitis.<br>• Appear as heterogeneous and non-liquid density of varying degrees in different locations.<br>• Does not have a definable wall.<br>• Necrosis can involve the pancreatic parenchyma and/or the peripancreatic tissues |
| WON (walled-off necrosis) | • Heterogeneous with liquid and non-liquid density with varying degrees of loculations<br>• Usually occurs 4 weeks after onset of necrotizing pancreatitis.<br>• Appear as an encapsulated collection in pancreatic and/or peripancreatic areas of necrosis.<br>• Contains a well-defined inflammatory wall. |

## Determinant based classification:

The primary highlight of the Determinant Based Classification was the introduction of the group called critical AP. This category and thereby the Determinant Based Classification stemmed from the results of a meta-analysis of 14 studies involving 1478 patients that evaluated the pooled effect of organ failure and infected pancreatic necrosis on mortality [13]. The meta-analysis demonstrated that mortality rate among patients who had both organ failure and infected pancreatic necrosis was 43%.


Severe Pancreatitis
Necrosis
Duodenum
Hemorrhage


Gallstones
Common bile duct
Gallstones in bile duct causing pancreatitis
Gallstones
Pancreatic duct
Duodenum

**Determinants**

The Determinant Based Classification primarily centers on causally associated factors (or determinants) for mortality. The determinants could be

1) local, i.e. (peri)pancreatic necrosis or
2) systemic, i.e. organ failure.

(Peri) pancreatic necrosis is defined as nonviable tissue located in the pancreas alone, or in the pancreas and peripancreatic tissues, or in the peripancreatic tissues alone. (Peri) pancreatic necrosis could be sterile or infected. Infected (peri) pancreatic necrosis is defined by the presences of either gas bubbles within necrotic areas on computed tomography, a positive culture of (peri) pancreatic necrosis obtained by image guided fine-needle aspiration, or positive culture of (peri) pancreatic necrosis obtained during the first drainage and/or necrosetomy. Organ failure is defined as a score of 2 or more according the Sequential Organ Failure Assessment (SOFA) system [(14)] ; or if there is a need for inotropic support and/or serum creatinine of > 2 mg/dl, and/or PaO2/FiO2 < 300 mm Hg. Organ failure for 48 h or more is defined as persistent, while it is defined as transient if less than 48 h.

The four categories in the Determinant Based Classification include:

**1)** mild - absence of both (peri) pancreatic necrosis and organ failure

2) moderate - sterile (peri) pancreatic necrosis and/or transient organ failure

3) severe - presence of either infected (peri) pancreatic necrosis or persistent organ failure

4) critical AP - presence of both infected (peri) pancreatic necrosis and persistent organ failure.

**CT – Image Acute Necrotising Pancreatitis**

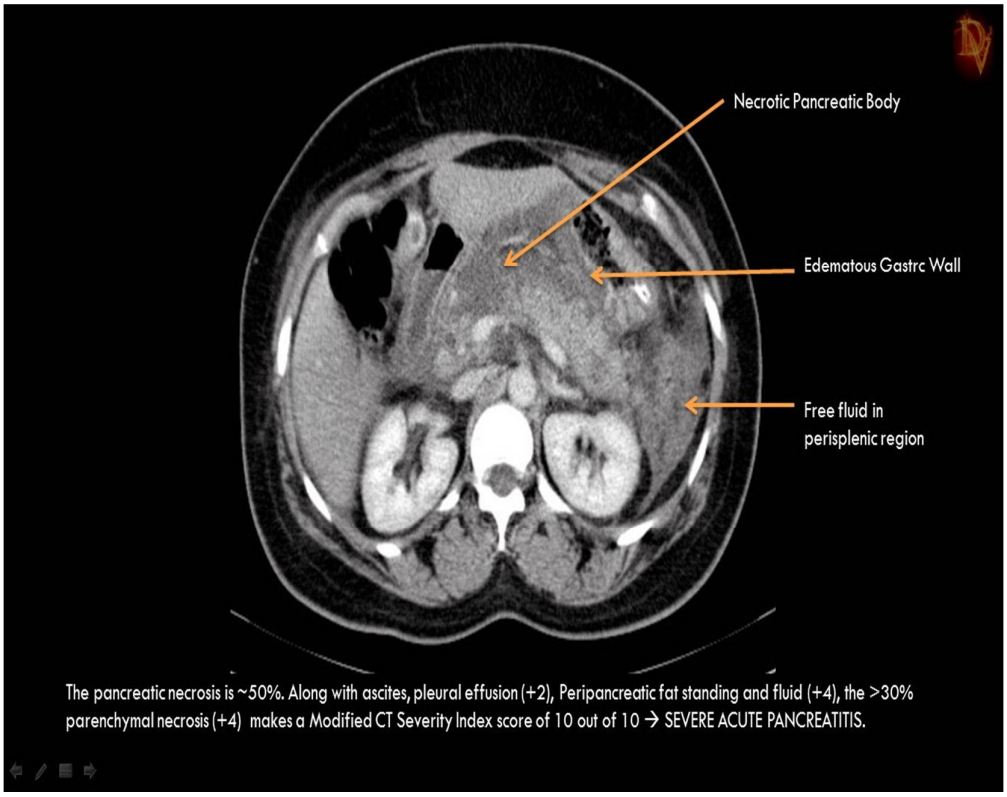


**Table 1: Definition of severity of acute pancreatitis according to different classification systems**

| Atlanta Classification | Revised Atlanta Classification | Determinant based classification |
|---|---|---|
| **Mild AP**<br>- *Minimal organ dysfunction and uneventful recovery*<br>- *Absence of organ failure and/or local complications*<br>**Severe AP**<br>- *Organ failure and/or local complications* | **Mild AP**<br>- *No organ failure*<br>- *No local or systemic complications*<br>**Moderately severe AP**<br>- *Transient organ failure AND/OR local or systemic complication OR exacerbation of pre-existing co-morbidities.*<br>**Severe AP**<br>- *Persistent organ failure (single or multiple)* | **Mild AP**<br>- *No organ failure*<br>- *No (peri)pancreatic necrosis*<br>**Moderate AP**<br>- *Sterile (peri)pancreatic necrosis AND/OR transient organ failure*<br>**Severe AP**<br>- *Infected (peri)pancreatic necrosis OR persistent organ failure*<br>**Critical AP**<br>- *Infected (peri)pancreatic necrosis AND persistent organ failure* |

## D. Etiology of Acute Pancreatitis :

Acute Pancreatitis is an inflammatory condition of the pancreas characterized clinically by abdominal pain and elevated levels of pancreatic enzymes in the blood. [15,16] In mild AP, the pancreas recovers its normal endocrine and exocrine functions and histology. Patients with severe acute pancreatitis develop permanent endocrine and exocrine insufficiencies depending on the extent of pancreatic injury and necrosis, irrespective of the etiology. Scarring of the pancreatic ducts can persists in some patients, mimicking the ductal changes of chronic pancreatitis. [17]

**FIG 3 PATHOPHYSIOLOGY OF PANCREATITIS**

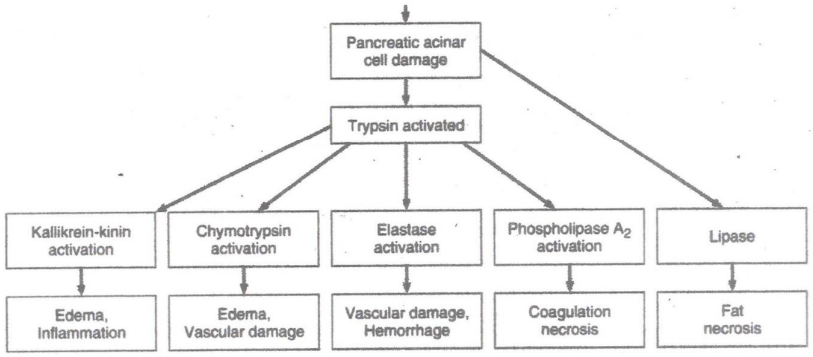



**Figure 15–3.** Hypothesized pathogenesis of acute pancreatitis. (Reproduced with permission from Marshall JB: Acute pancreatitis: A review with an emphasis on new developments. Arch Intern Med 1993;153:1185.)

The complex pathogenesis of AP is still not very clearly understood, although several theories based on animal model studies have been put forward. A number of conditions are suspected, with varying degrees of certainty, to induce acute pancreatitis. Gallstones and chronic alcohol abuse accounts for 75-80% of the cases, the rest are attributed to other factors such as Hypertriglyceridemia, Hypercalcemia, drugs, smoking, genetic mutations, infection, trauma, factors causing ampullary obstruction etc. A minor subset of patients have no identifiable etiology, and the cause of pancreatitis remains undetermined. As our knowledge and understanding of the disease grows, the number of so called "idiopathic" cases will decrease as our list of causes grows.

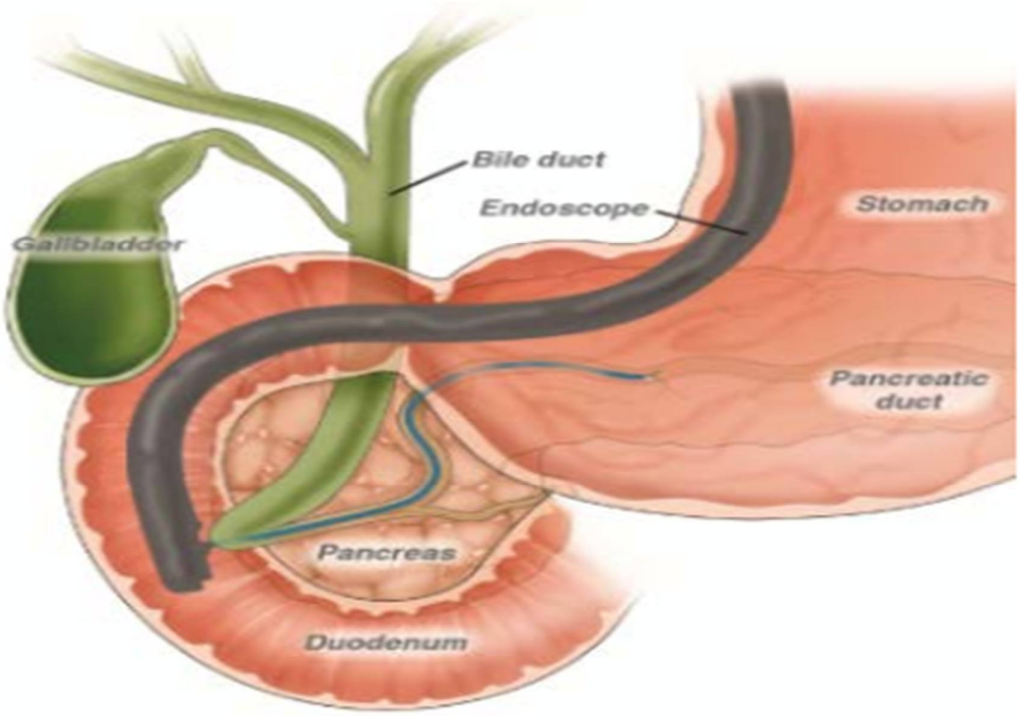
A review on the epidemiology of acute pancreatitis found an increasing incidence (especially that due to alcohol and gallstones in some areas) and decreasing case-fatality rate[18]

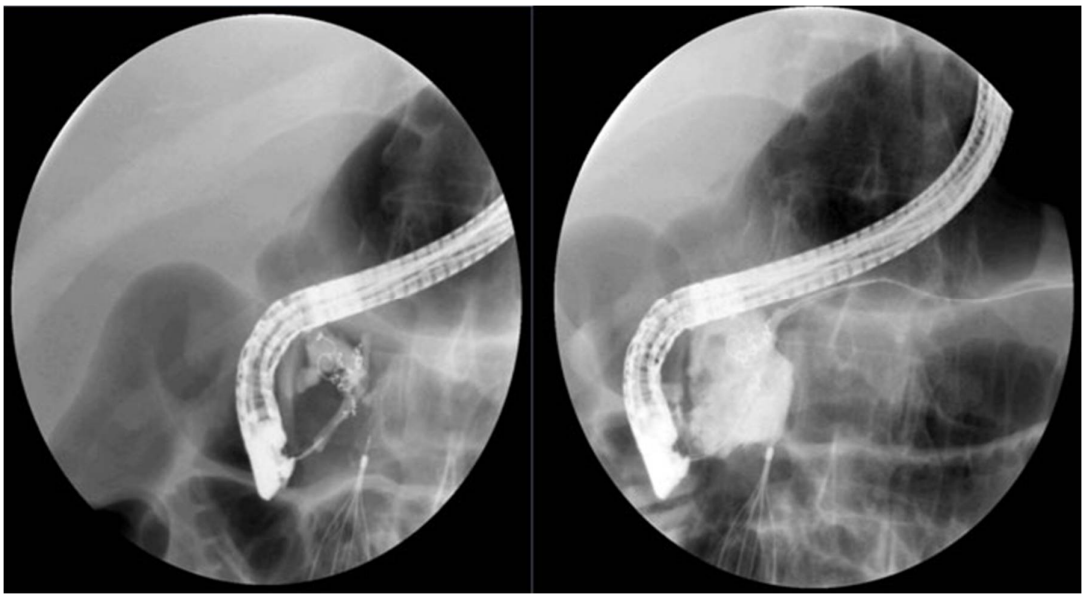
**Gallstones and other causes of mechanical ampullary obstruction:**

Mechanical ampullary obstruction can be induced by gallstones and a variety of disorders. The most common cause of acute pancreatitis in most areas of the world is gallstones (including microlithiasis), which accounts for 35 to 40 % of cases.[19] Cholecystectomy and clearing the common bile duct of stones prevents recurrence, confirming the cause and effect relationship. [20]

The mechanism by which the passage of gallstones induces pancreatitis is unknown. Two factors have been suggested as the possible initiating event in gallstones pancreatitis:

a) Reflux of bile into the pancreatic duct due to transient obstruction of the ampulla during passage of gallstones[21]
b) Obstruction at the ampulla secondary to stones or edema resulting from the passage of gallstones[22]


Bile duct
Endoscope
Stomach
Gallbladder
Pancreatic duct
Pancreas
Duodenum



**Gallstone Ultra sonogram**

**Incidence of gallstones (biliary pancreatitis):**

Although 35 percent of attacks of Acute Pancreatitis are caused by gallstones, only 3 to 7 % of patients with gallstones eventually develop pancreatitis. [20,23] Gender and stone size may be risk factors for gallstone pancreatitis. The risk of developing AP in patients with gallstones is greater in men; however, more women develop this disorder since gallstones occur with increased frequency in women.

It has been suggested that acute pancreatitis is associated with a stone diameter of less than 5mm and that smaller stones or microlithiasis

are more likely than larger stones to pass through the cystic duct and cause obstruction at the ampulla. [24,25]

**Management of Biliary Pancreatitis**

The diagnosis of gallstones pancreatitis should be suspected if the patient has a prior history of biliary colic. In addition all patients with a first attack of acute pancreatitis should have an abdominal ultrasound to search for gallstones, common bile duct stones or signs of extrahepatic biliary tract obstruction. [23] Laboratory values obtained during the acute attack may also assist in making the diagnosis. Serum alanine

aminotransferase (ALT) concentration is the most clinically useful parameter in predicting a gallstone etiology in patients with acute pancreatitis. [26]

If gallstone pancreatitis is suspected on the basis of imaging and laboratory findings and there is no history of alcohol abuse, cholecystectomy with cholangiography is recommended during the same hospitalization. ERCP is performed after laproscopic cholecystectomy if a common bile duct stone is found and not removed at surgery.

**Biliary sludge and Microlithiasis**

Biliary sludge is a viscous suspension in the gallbladder bile that may contain small stones (<5 mm in diameter). It is formed by modification of hepatic bile by gallbladder mucosa; thus hepatic bile samples may be insufficient for its diagnosis.[27]

Most patients with biliary sludge are asymptomatic. [27] Sludge appears as a mobile, low- amplitude echo on ultrasound that layers in the most dependent part of the gallbladder and is not associated with shadowing. Microscopic analysis of bile in patients with sludge often shows cholesterol monohydrate crystals or calcium bilirubinate granules. [28]

Sludge is typically found in patients with functional or mechanical bile stasis, such as those undergoing a prolonged fast, with distal bile duct obstruction, or on total parenteral nutrition. In addition, certain drugs that excreted by hepatocytes, such as ceftriaxone, can complex with bile to form sludge within the biliary system when its solubility in bile is exceeded.

Biliary sludge is commonly found in patients with acute pancreatitis with no obvious cause. However, the association between biliary sludge and acute pancreatitis is unproven. Because of the high risk of recurrence, cholecystectomy is recommended in patients who have had an episode of pancreatitis and have biliary sludge.[29]

**Other causes of Acute Pancreatitis secondary to Ampullary Obstruction**

Other conditions causing obstruction of the ampulla that have been associated with pancreatitis include biliary ascariasis, periampullary diverticula and pancreatic and periampullarytumours. Intraductal papillary mucinous neoplasms (IPMN) of the pancreas are being recognized increasingly and may occasionally present as acute pancreatitis, especially in elderly nonalcoholic males.

## Alcohol and Acute Pancreatitis

Acute alcoholic pancreatitis, along with gallstones pancreatitis, is the most common diagnosis among patients hospitalized with pancreatic disease.[30-34] Approximately 10 % of chronic alcoholics develop attacks of clinically acute pancreatitis. Studies have shown that not all patients progress to chronic pancreatitis, even with continued alcohol abuse. [35]

Why only a small proportion of all alcoholics develop pancreatitis, what genetic and environmental factors influence the development of pancreatitis in alcoholics, and what is the exact mechanism of pancreatic injury by alcohol remain unanswered in spite of extensive and ongoing research.

## Role of smoking in the etiology of Acute Pancreatitis

Until recently, smoking was thought to be a risk factor due to its association with alcohol. However, at least three large studies have suggested that cigarette smoking is an independent risk factor for acute pancreatitis by mechanisms that are unclear. [36,37,38]

## Hypertriglyceridemia

Serum triglyceride concentrations above 1000 mg/dL can precipitate attacks of acute pancreatitis, although the pathogenesis of

inflammation in this setting is unclear.[39] Hypertriglyceridemia may account for 1.3 to 3.8 percent of cases of acute pancreatitis. [40]

Hypertriglyceridemia, with concentrations severe enough to trigger attacks of acute pancreatitis, may even be present in children as a part of a spectrum of inherited lipoprotein metabolism disorders. Acquired causes of hypertriglyceridemia include obesity, diabetes mellitus, hypothyroidism, pregnancy, estrogen or tamoxifen therapy, glucocorticoid excess, nephrotic syndrome and beta blockers.

**Hypercalcemia**

Hypercalcemia of any cause can lead to acute pancreatitis, although actual incidence is low. Proposed mechanisms include deposition of calcium in the pancreatic duct and calcium activation of trypsinogen within the pancreatic parenchyma. [41]

**Genetic mutations**

There have been tremendous advances in the past few years in our understanding of the genetic basis of acute pancreatitis. Some genetic disorders are associated with a high-penetrance (eg, mutations at codons 29 and 122 of the serine protease I (cationic trypsinogen) gene (PRSS1), while others have a low penetrance and are more frequent in the general population (eg, mutations in the serine protease inhibitor Kazal type I

(SPINK1), which may act as a disease modifier). In addition, certain mutations in the cystic fibrosis gene (CFTR) have been associated with acute pancreatitis.

Inherited forms of pancreatitis, which may present as recurrent acute pancreatitis but eventually progresses to chronic pancreatitis, may be inherited as autosomal dominant, autosomal recessive or be a mutagenic disorder as a result of mutations in these or yet unidentified genes.

**Drug induced Acute Pancreatitis**

Acute pancreatitis related to drug or medication use is uncommon. The literature on drug-induced pancreatitis mostly consists of case reports and anecdotal account. Many drugs have been implicated as etiologic agents, and the list continues to grow. [42]

The following drugs were definitely associated with pancreatitis by at least two of the three reviews of this subject [42]:

- AIDS therapy-didanosine, pentamidine
- Antimicrobial agents-metronidazole, stibogluconate, sulfonamides, tetracycline
- Diuretics-furosemide, thiazides
- Drugs used for inflammatory bowel disease-sulfasalazine, 5-ASA

- Immunosuppressive agents –L-asparaginase, azathioprine
- Neuropsychiatric agents-valporic acid
- Anti-inflammatory drugs-sulindac, salicylates
- Others-calcium, estrogen,tamoxifen

The pathogenesis of drug-induced pancreatitis may be due to an idiosyncratic response in some cases (eg, 6-mercaptopurine, aminosalicylates, and sulfonamides) or due to a direct toxic effect (eg, diuretics, sulfonamides).

A high index of suspicion and careful drug history are essential for making the diagnosis. The time course of developing the disorder depends upon the drug involved.

**Infectious causes of Acute Pancreatitis**

There are numerous case reports of acute pancreatitis due to a wide variety of infectious agents.

Cases of definite pancreatitis were associated with the following organisms[(43)]:

- Viruses–mumps, Coxsackie virus, Hepatitis B, Cytomegalo virus, Varicella-zoster, Herpes simplex

- Bacteria-Mycoplasma, legionella, Leptospira, Salmonella
- Fungi-Aspergillus
- Parasites-Toxoplasma, Cryptosporidium, Ascaris

The frequency with which these infections lead to pancreatitis is not known.

**Trauma**

Blunt or penetrating trauma can damage the pancreas and cause acute pancreatitis, although these injuries are uncommon due to the retroperitoneal location of the gland. The diagnosis of traumatic pancreatitis is difficult and requires a high degree of suspicion.

**Vascular disease**

Pancreatic ischemia is an uncommon cause of clinically significant pancreatitis. However, ischemia with resultant pancreatitis has been reported in the following circumstances[45]:

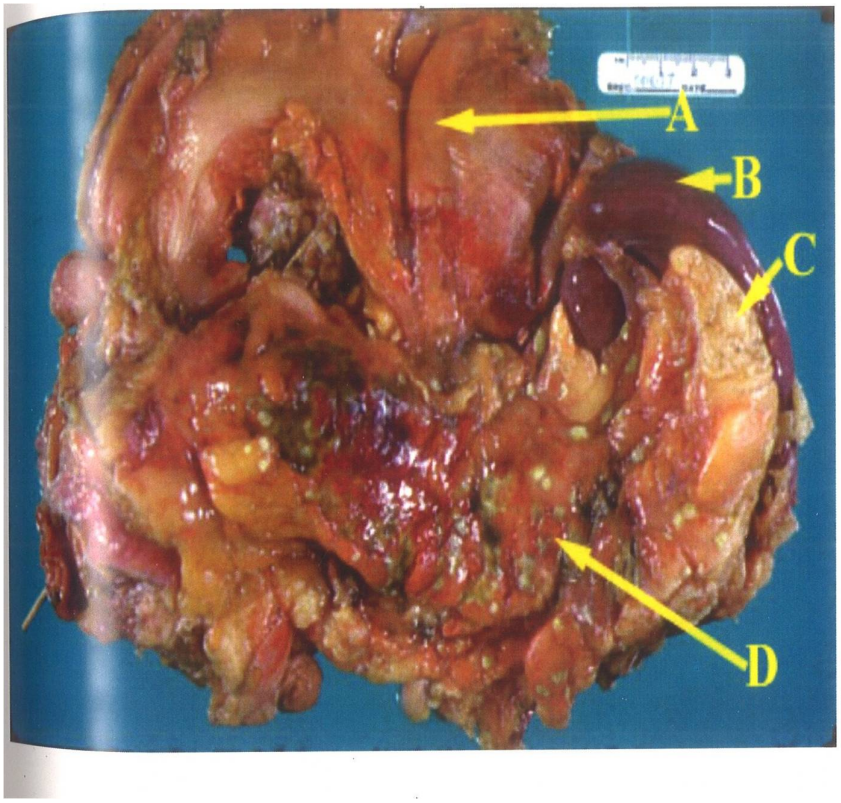
- Vasculitis (SLE and PAN)
- Atheroembolism
- Intraoperative hypotension
- Hemorrhagic shock

## Acute Pancreatitis Post-ERCP

Asymptomatic hyperamylasemia occurs in 35 to 70 percent of patients undergoing ERCP. A diagnosis of post-ERCP pancreatitis is generally made if the hyperamylasemia is accompanied by persistent severe upper abdominal pain, often with nausea and vomiting. Acute pancreatitis occurs in about 3 % of patients undergoing diagnostic ERCP, 5% of patients undergoing therapeutic ERCP, and up to 25 % undergoing sphincter of Oddimanometric studies. [46]

## Idiopathic Acute Pancreatitis

No obvious etiology is identifiable [history (e.g., alcohol, family history), laboratory tests (e.g., gallstone pancreatitis, hyperlipidemia, hypercalcemia), and abdominal ultrasound] in up to 30 % of patients with acute pancreatitis. The term idiopathic pancreatitis is used for these who have no cause found even after an exhaustive search for an etiology. Most recommendations suggest that extensive investigation for unusual cause of pancreatitis is not required after the first episode of unexplained pancreatitis. Approximately 15 to 25 % of patients with acute pancreatitis may be labelled as idiopathic and this figure will probably decrease in the future with better identification of different causes.




**E. Pathogenesis of acute pancreatitis:**

Acute pancreatitis is an inflammatory condition of the pancreas characterized clinically by abdominal pain and elevated levels of pancreatic enzymes in the blood. The disease process can be localized or affect the whole pancreas, and is a disease with typically unpredictable course.[47] A number of conditions known to induce this disorder. The exact mechanisms by which diverse etiological factors induce an attack are still unclear. It is generally believed that the earliest events in acute pancreatitis occur within acinar cells. Acinar cell injury early in acute pancreatitis leads to a local inflammatory reaction. If this inflammatory reaction is marked, it leads to a systemic inflammatory response

syndrome (SIRS). An excessive SIRS leads to distant organ damage and multiple organ dysfunction syndrome (MODS).MODS associated with acute pancreatitis is the primary cause of morbidity and mortality in this condition. [48]

**Animal Models Demonstrate Pathogenesis of Inflammatory Response**

Although a number of animal models have been developed to understand the pathogenesis of acute pancreatitis, none is strictly comparable to the human condition.[49] Alcohol abuse and gall stones are implicated in the etiopathogenesis of 90% of cases of acute pancreatitis in India [50] which is comparable to their role stated in western literature. [33,34,51-53]

However, none of the existing animal models duplicates these situations. In addition, the commonly used agents for inducing pancreatitis in animal models, such as cerulin and a choline-deficient ethionine-supplemented diet, are not recognized causes of human acute pancreatitis. [49]

Nevertheless, the structural and biochemical changes seen in early phases of acute pancreatitis are remarkably constant in different animal models, and similar changes have been demonstrated in human acute

pancreatitis. Furthermore, the clinical and pathologic features of human acute pancreatitis, regardless of the inciting event, are very similar.[49,50,51]

Despite the limitations of animal models, the data suggest that a similar cascade of events occurs once pancreatitis begins independent of the inciting event or initial mechanism. [50] Animal studies have shown that this cascade cannot be halted successfully unless therapy is initiated either prophylactically or within a few hours of the initiating event. It is not clear from these studies why some individuals develop interstitial or edematous pancreatitis, while others go on to develop the necrotizing form of the disease. [52]

Thus animal models help us understand the mechanisms and subsequent consequences of intra-pancreatic digestive enzyme activation, the generation and role of cytokines and other inflammatory mediators in the pancreatic acinar cell, and the role of extra-acinar players such as inflammatory cells in pancreatic inflammation. Further, mechanistic advances have also been made in understanding the modes of cell death, including apoptosis and necrosis, and their relevance to pancreatitis.

From these experimental studies, numerous factors have been implicated in causing pancreatic injury, including intra-pancreatic digestive enzyme activation [53], cytokines and chemokines[54], inflammatory cells [55], peptide mediators such as substance P [56], small

molecule mediators such as nitric oxide [57], reactive oxygen species [43], polyamine depletion [58], and cyclooxygenase (COX) – 2 [59]. While pancreatitis may be due to several of these factors acting in different ways, the disease frequently develops in severity over time and thus it is important to understand the initial events that trigger or exacerbate it, so as to design treatments that are beneficial if administered in the early stages of presentation.

**Triggering event:**

Only a small percent of people with predisposing factors actually develop acute pancreatitis. For example, several large population based studies have drawn the conclusion that only 3-7 % with gallstones, 10% of alcoholics and <1% of patients with hypercalcemia eventually contract pancreatitis. [60]

The exact mechanism of induction of acute pancreatitis is not known. However several theories have been put forward.

In alcohol induced acute pancreatitis ongoing studies are being conducted to elucidate the following possible triggering pathways [30]:

1) sensitization of acinar cells to CCK by the activation of zymogens
2) Potentiation of the effect of CCK on the activation of transcription factors, nuclear factor kB, and activating protein – 1

3) Generation of toxic metabolites such as acetaldehyde and fatty acid ethyl esters
4) Sensitization of the pancreas to the toxic effects of coxsackie virus B3
5) Activation of pancreatic stellate cells by acetaldehyde and oxidative stress and subsequent increased production of collagen and other matrix proteins.

Two factors have been suggested as the possible initiating events in gallstones pancreatitis:

1) reflux of bile into the pancreatic duct due to transient obstruction of the ampulla during passage of gallstones
2) obstruction at the ampulla secondary to stone(s) or edema resulting from the passage of a stone

In hyperlipidemia it has been suggested that the triggering factor is free fatty acids that are released from serum triglycerides in toxic concentrations by the action of pancreatic lipase within pancreatic capillaries. [39]

## Early Acute Changes

### Intra-acinar activation of proteolytic enzymes:

One of the earliest events seen in different models of acute pancreatitis is blockade of secretion of pancreatic enzymes while synthesis continues.

The central requirement for induction of acute pancreatitis is intraacinar activation of these proteolytic enzymes, which ultimately leads to autodigestive injury to the gland.

A proposed mechanism by which intra-acinar activation occurs and leads to pancreatic destruction in animal models of pancreatitis is as follows: [60]

1) a devastating event occurs very early which allows generation of large amounts of active trypsin within the pancreas.
2) Collection of lysosomal enzymes such as cathepsin B and digestive vacuoles within the acinar cell/ in the normal acinar cell, these two groups of enzymes are carefully sorted by the Golgi network.
3) In early pancreatitis, however, cathepsin B cleaves the trypsinogen activation peptide from trypsinogen within the acinar vacuoles, leading to intra pancreatic activation of trypsin.

4) The normal defense mechanisms of the pancreas are overwhelmed by the large amounts of trypsin released.
5) The intra pancreatic release of trypsin leads to activation of more trypsin, and other pancreatic enzymes such as phospholipase, chymotrypsin, and elastase.
6) Trypsin also activates other enzymes cascades including complement, kallikrein- kinin, coagulation and fibrinolysis.
7) The intra pancreatic release of active pancreatic enzymes leads to pancreatic autodigestion

This sets up a vicious cycle of active enzymes damaging cells, which then release more active enzymes. The destruction spreads along the gland and into the peripancreatic tissue.

**Microcirculation injury:**

The release of pancreatic enzymes damage the vascular endothelium and the interstitium as well as the acinar cells. Microcirculatory changes, including vasoconstriction, capillary stasis, decreased oxygen saturation and progressive ischemia, occur early in experimental models of acute pancreatitis. These changes lead to increased vascular permeability and swelling of the gland (edematous or interstitial pancreatitis). Vascular injury could lead to local microcirculatory failure and amplification of the pancreatic injury. [45, 61]

There is also speculation about the role of ischemia- reperfusion injury in the pancreas. Reperfusion of damaged tissues leads to the release of free radicals and inflammatory cytokines into the circulation, which could cause further injury.[61] The importance of microcirculatory injury can be appreciated by the importance of aggressive fluid replacement in the management of acute pancreatitis, which minimizes this injury.

**Leucocyte chemoattraction and release of cytokines**

Microscopic and radionuclide studies using Indium – 111 tagged leukocytes show marked glandular invasion by macrophages and polymorphonuclear leukocytes in early stages of animal and human pancreatitis. Activation of complement and the subsequent release of C5a have a significant role in the recruitment of these inflammatory cells.

Granulocyte and macrophage activation causes the release of proinflammatory cytokines (tumor necrosis factor, interleukins 1,6 and 8), arachidonic acid metabolites (prostaglandins, platelet-activating factor, and leukotrienes), proteolytic and lipolytic enzymes, and reactive oxygen metabolites which overwhelm the scavenging capacity of endogenous antioxidant systems. These substances also interact with the pancreatic microcirculation to increase vascular permeability and induce thrombosis and hemorrhage, leading to pancreatic necrosis.

Activated pancreatic enzymes, microcirculatory impairment, and the release of inflammatory mediators lead to rapid worsening of pancreatic damage and necrosis. This interaction makes it difficult to estimate the individual roles of these factors in inducing pancreatic damage. In addition, approximately 80 percent with pancreatitis develop only interstitial pancreatitis rather than necrotizing pancreatitis; the factors involved in limiting the pancreatic damage are not well understood.

**Systemic Response**

Some patients with severe pancreatic damage develop systemic complications including fever, acute respiratory distress syndrome (ARDS), pleural effusions, renal failure, shock, and myocardial depression.

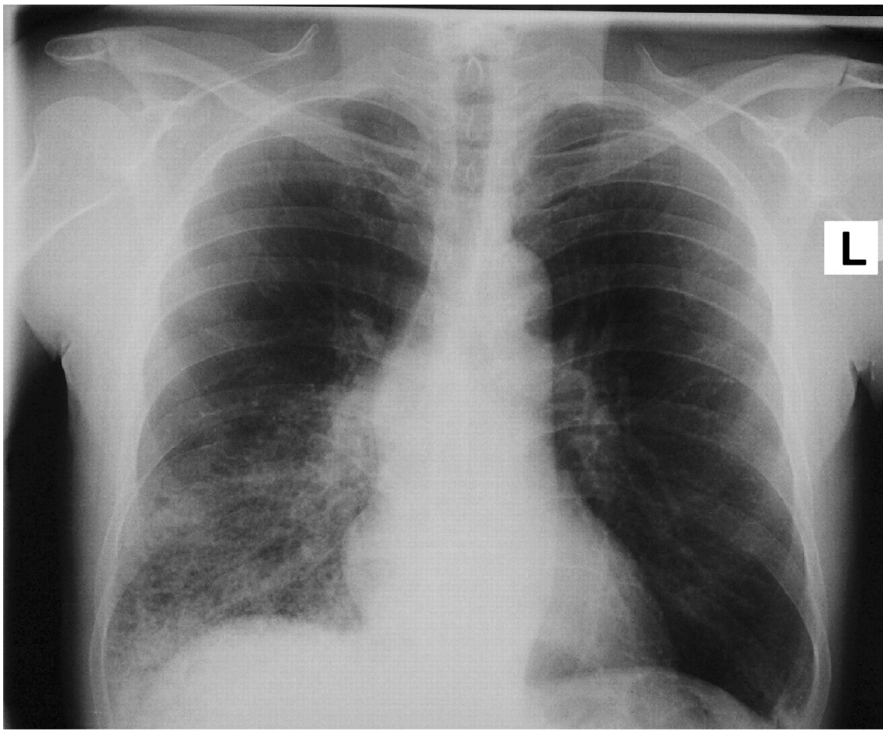
This systemic inflammatory response syndrome (SIRS) is probably mediated by activated pancreatic enzymes (phospholipase, elastase, trypsin, etc) and cytokines (tumor necrosis factor, platelet activating factor) released into the circulation from the inflamed pancreas.

ARDS, in addition to being secondary to microvascular thrombosis, may be induced by active phospholipase A ( lecithinase), which digests lecithin, a major component of surfactant. Myocardial

depression and shock are thought to be secondary to vasoactive peptides and a myocardial depressant factor. Acute renal failure has been explained on the basis of hypovolemia and hypotension. Metabolic complications include hypocalcemia, hyperlipidemia, hyperglycemia, hypoglycemia, and diabetic ketoacidosis. The pathogenesis of hypocalcemia is multifactorial and includes calcium- soap formation, hormonal imbalances (eg, pararthyroid hormone, calcitonin, glucagon), binding of calcium by free fatty acid-albumin complexes, and intracellular translocation of calcium.

These systemic complications are uncommon and much less severe in patients with interstitial pancreatitis than in those with necrotizing pancreatitis. However, only about 50 percent of patients with necrotizing pancreatitis develop organ failure, and this complication cannot be predicted from the degree of pancreatic necrosis. One study suggested that an increased tissue concentration of macrophage migratory inhibitory factor was a critical factor in the pathogenesis of severe acute pancreatitis.

**Acute Respiratory Distress syndrome**




Bacterial translocation – The normal human gut prevents the translocation of bacteria into the systemic circulation through a complex barrier that consists of immunologic, bacteriologic, and morphologic components. During the course of acute pancreatitis, the gut barrier is compromised, leading to translocation of bacteria, which can result in local and systemic infection. The breakdown in the gut barrier is thought to be a consequence of ischemia due to hypovolemia and pancreatitis-induced gut arteriovenous shunting.

Most infections in acute pancreatitis are caused by common enteric organisms suggesting that they originate from the gastrointestinal tract.

The consequences of bacterial translocation the gut in acute pancreatitis can be lethal. Local bacterial infection of pancreatic and peripancreatic tissues occurs in approximately 30 percent of patients with severe acute pancreatitis, potentially resulting in multiorgan failure and its sequelae.

**The Inflammatory Cascade in Acute Pancreatitis:**

**Triggering events:**

A number of situations can precipitate acute pancreatitis in humans, but only a small fraction of patients with these predisposing factors ultimately develop the disease. For example, only 3 to 7 percent of people with gallstones [(23)]; 10 percent of alcoholics; and a few patients with hypercalcemia[(60)] eventually develop acute pancreatitis.

**Alcoholic Pancreatitis**

The exact mechanism of induction of pancreatitis by these agents in not known. It is unclear, for example, why alcohol- induced pancreatitis occurs only after manyyears of alcohol abuse and not after a single binge in humans not habituated to alcohol use. However, several mechanisms have been proposed for the development of acute pancreatitis in alcoholics:[(30)]

1) Sensitization of acinar cells to CCK – induced premature activation of zymogens
2) Potentiation of the effect of CCK on the activation of transcription factor, nuclear factor kB, and activating protein – 1
3) Generation of toxic metabolites such as acetaldehyde and fatty acid ethyl esters
4) Sensitization of the pancreas to the toxic effects of coxsackie virus B3
5) Activation of pancreatic stellate cells by acetaldehyde and oxidative stress and subsequent increased production of collagen and other matrix proteins.

Different mechanisms have been proposed for other forms of pancreatitis.

**Gallstones Pancreatitis**

**1)** reflux of bile into the pancreatic duct due to transient obstruction of the ampulla during passage of gallstones [47]

**2)** obstruction at the ampulla secondary to stone(s) or edema resulting from the passage of a stone [48]

**3)** Another defense mechanism involves mesotrypsin and enzyme Y, which lyses and inactivates trypsin.

**4)** non specific antiproteases such as alpha – 1 antitrypsin and alpha – 2 macroglobulin are present in the pancreatic interstitium.

To summarize, cationic trypsin is the most abundant form of trypsin produced by the pancreas and is the primary catalyst for the conversion of pancreatic zymogens into pancreatic digestive enzymes after they are secreted into the duodenum. Premature activation of digestive enzymes in the pancreas is the major cause of pancreatic injury and immune system activation, leading to acute pancreatitis and later chronic pancreatitis. The primary defense against pancreatitis is to control trypsin activity, either through prevention of premature activation of trypsinogen to trypsin, or by the destruction, inhibition, or elimination of trypsin from the pancreas. [63]

**Intra- acinar activation of proteolytic enzymes**

One of the earliest events in the different models of acute pancreatitis is blockade of secretion of pancreatic enzymes while synthesis continues.[90] It is becoming increasingly clear that the central event in the triggering of acute pancreatitis is the activation of these proteolytic enzymes within the acinar cells of the gland leading to auto digestive injury of pancreatic tissue

**Proposed cascade of events in Intra-acinar activation of proteolytic enzymes:**

- Co localization of lysosomal enzymes, such as cathepsin B and digestive enzymes including trypsinogen, occurs in unstable vacuoles within the acinar cell [(64)]. In the normal acinar cell, the golgi network carefully sorts these two groups of enzymes. In early pancreatitis, however, cathepsin B cleaves the trypsinogen activation peptide from trypsinogen within the acinar vacuoles, leading to intrahepatic activation of trypsin [(64)]
- The normal defense mechanisms of the pancreas are overwhelmed by the large amounts of trypsin released. In addition, the intrapancreatic release of trypsin leads to activation of more trypsin, and other pancreatic enzymes such as phospholipase, chymotrypsin, and elastase. Trypsin also activates other enzyme cascades including complement, kallikrein – kinin, coagulation and fibrinolysis. [(29)]
- The intrapancreatic release of active pancreatic enzymes leads to pancreatic autodigestion, setting up a vicious cycle of active enzymes damaging cells, which then release more enzymes. The destruction spreads along the gland into the peripancreatictissue[(65)]

The activation of trypsinogen occurs before either biochemical or morphological injury to acinar cell is evident.

An in vitro model found that complete inhibition of pancreatic cathepsin B activity with E-644(a specific potent and irreversible cathepsin B inhibitor) prevented cerulean-induced trypsinogen activation[66]

This observation supports the significance of cathepsin B activation of trypsinogen, and the importance of co-localization of pancreatic digestive enzymes and lysosomal hydrolases. In addition, it suggest that complete inhibition of cathepsin B may be of benefit in either the prevention or treatment of acute pancreatitis

Other mechanism besides cathepsin B have also been suggested to have a role like trypsinogenautoactivation or activation by other lysosomal proteinases

**Microcirculation injury:**

The release of pancreatic enzymes damage the vascular endothelium and the interstitium as well as the acinar cells. Microcirculatory changes, including vasoconstriction, capillary stasis, decreased oxygen saturation ,and progressive ischemia, occur early in experimental models of acute pancreatitis. These changes lead to increased vascular permeability and

swelling of the gland (edematous or interstitial pancreatitis). Vascular injury could lead to local microcirculatory failure and amplification of the pancreatic injury. [19,37]

There is also speculation about the role of ischemia- reperfusion injury in the pancreas. Reperfusion of damaged tissues leads to the release of free radicals and inflammatory cytokines into the circulation, which could cause further injury.[37] The importance of microcirculatory injury can be appreciated by the importance of aggressive fluid replacement in the management of acute pancreatitis, which minimizes this injury.

**Release of inflammatory mediators**

Microscopic and radionuclide studies using Indium – 111 tagged leukocytes show marked glandular invasion by macrophages and polymorphonuclear leukocytes in early stages of animal and human pancreatitis. Activation of complement and the subsequent release of C5a have a significant role in the recruitment of these inflammatory cells.

Granulocyte and macrophage activation causes the release of proinflammatory cytokines (tumor necrosis factor, interleukins 1,6 and 8), arachidonic acid metabolites (prostaglandins, platelet-activating factor, and leukotrienes), proteolytic and lipolytic enzymes, and reactive

oxygen metabolites which overwhelm the scavenging capacity of endogenous antioxidant systems. These substances also interact with the pancreatic microcirculation to increase vascular permeability and induce thrombosis and hemorrhage, leading to pancreatic necrosis.

Activated pancreatic enzymes, microcirculatory impairment, and the release of inflammatory mediators lead to rapid worsening of pancreatic damage and necrosis. This interaction makes it difficult to estimate the individual roles of these factors in inducing pancreatic damage. In addition, approximately 80 percent with pancreatitis develop only interstitial pancreatitis rather than necrotizing pancreatitis; the factors involved in limiting the pancreatic damage are not well understood

**Complications**

Pancreatitis can cause serious complications, including:

**Pancreatic pseudocysts** are common sequelae of acute pancreatitis or chronic pancreatitis, and the most common cystic lesion of the pancreas. They are important both in terms of management and differentiation from other cystic processes or masses in this region.

## Terminology

The following are the latest terms according to the updated Atlanta classification to describe fluid collections associated with acute pancreatitis 10,11:

- fluid collections in interstitial oedematous pancreatitis
  - acute peripancreatic fluid collections (APFC): in the first 4 weeks: non-encapsulated peripancreatic fluid collections
  - pseudocysts: develop after 4 weeks; encapsulated peripancreatic or remote fluid collections
- fluid collections in necrotising pancreatitis
  - acute necrotic collections (ANCs): in the first 4 weeks; non-encapsulated heterogeneous non-liquefied material
  - walled-off necrosis (WON or WOPN): develop after 4 weeks; encapsulated heterogeneous non-liquefied material

## Clinical presentation

Pancreatic pseudocysts are frequently found on imaging follow-up of pancreatitis, and may in themselves be asymptomatic for some time. Presentations attributable to a pseudocyst include:

- mass effect
  - biliary obstruction
  - gastric outlet obstruction
- secondary infection

**Pathology**

Pseudocysts occur from disruption of pancreatic duct structure with resulting leakage and accumulation of pancreatic juice resulting in haemorrhagic fat necrosis. A severe inflammatory reaction that is incited by this results in encapsulation of the cyst by fibrous tissue. This usually takes 4-6 weeks. In approximately 50% of cases the cyst retains a communication with the pancreatic duct  Such cysts are more problematic to treat, and are more likely to recur.

**Aetiology**

- acute or chronic pancreatitis (most common)
- pancreatic trauma
- iatrogenic, e.g. post partial gastrectomy

**Radiographic features**

Pseudocysts are fluid filled oval or round collections with a relatively thick wall. They can be multiple and are most commonly

located in the pancreatic bed. However, they can be found anywhere from the groin to the mediastinum and even in the neck, having ascended in the retroperitoneum via the diaphragmatic hiatuses into the mediastinum 5.

It is not possible to reliably distinguish infected from non-infected pseudocysts on imaging alone .

**Ultrasound**

Hypoechoic or anechoic collections with low-level echoes are often seen dependently representing debris.

**CT**

Pseudocysts appear as well-circumscribed, usually round or oval peripancreatic fluid collections of homogeneously low attenuation, that are usually surrounded by a well-defined enhancing wall .

According to the revised Atlanta classification, pseudocysts contain no non-liquefied components within the fluid collection .

**MRI**

- **T1**
  - hypo-intense (fluid signal) centre
  - wall demonstrates mild early enhancement, which progressively becomes more intense
- **T2**
  - hyperintense (fluid signal)
  - layering or dependent debris, highly specific

**Treatment and prognosis**

Treatment of pseudocysts depends on size and presence of superimposed infection, as well as local mass effect (usually related to size). If the cysts are small (less than 4-6 cm) they mostly resolve

spontaneously. Approximately half of all pseudocysts resolve spontaneously . Indications for drainage include

- infection
- large size: > 4-6 cm
- mass effect
  - gastric outlet obstruction
  - hydronephrosis
  - biliary obstruction
- growth on serial scanning

Treatment options include:

- open surgical debridement, or cystenterostomy with a Roux-en-Y jejunal loop
- endoscopic drainage into the stomach (or duodenum)
- percutaneous drainage
  - remains somewhat controversial, although increasingly accepted
  - many centres report high safety and efficacy
  - critics raise concern regarding potential reaccumulation and fistula formation to the skin (especially in patients with severe pancreatitis)

- Octreotide infusion: decreases amount of pancreatic secretions

Cysts that do not communicate with the pancreatic duct usually do not recur, and are unlikely to create fistulae

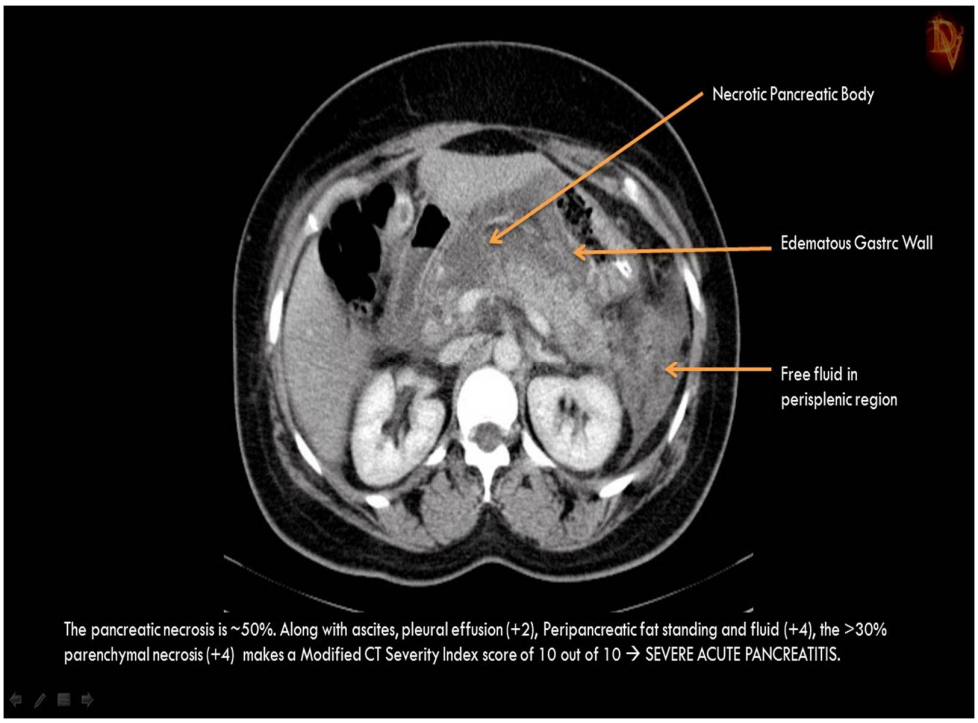
- **Infection.** Acute pancreatitis can make your pancreas vulnerable to bacteria and infection. Pancreatic infections are serious and require intensive treatment, such as surgery to remove the infected tissue.
- **Kidney failure.** Acute pancreatitis may cause kidney failure, which can be treated with dialysis if the kidney failure is severe and persistent.
- **Breathing problems.** Acute pancreatitis can cause chemical changes in your body that affect your lung function, causing the level of oxygen in your blood to fall to dangerously low levels.
- **Diabetes.** Damage to insulin-producing cells in your pancreas from chronic pancreatitis can lead to diabetes, a disease that affects the way your body uses blood sugar.
- **Malnutrition.** Both acute and chronic pancreatitis can cause your pancreas to produce fewer of the enzymes that are needed to break down and process nutrients from the food you eat. This can lead to malnutrition, diarrhea and weight loss, even though you may be eating the same foods or the same amount of food.

**Pancreatic cancer.** Long-standing inflammation in your pancreas caused by chronic pancreatitis is a risk factor for developing pancreatic cancer

**F. Management of Acute pancreatitis :**

Acute pancreatitis can be divided into two broad categories:

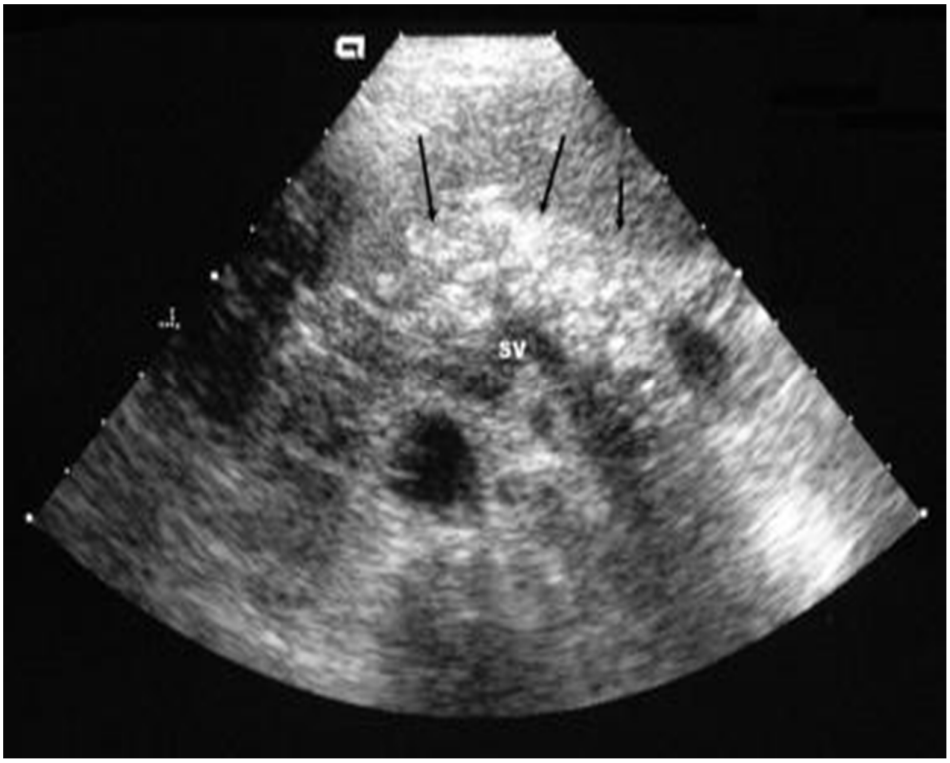
1) Edematous or mild acute pancreatitis and
2) Necrotizing or severe acute pancreatitis




Treatment of acute pancreatitis is based upon the severity of the condition, which is determined by the clinical, laboratory and a severity scoring system. Most attacks of acute pancreatitis are mild with recovery occurring within five to seven days. Death is unusual in such patients. In contrast, severe necrotizing pancreatitis is associated with a high rate of

complications and significant mortality. One study characterized an intermediate group called “moderately severe acute pancreatitis”, which comprised patients with local complications but no organ failure. [10] These patients had low mortality like mild acute pancreatitis but morbidity (requiring prolonged hospital stay and interventions) like severe acute pancreatitis. A subgroup of patients has early severe acute pancreatitis characterized by extended pancreatic necrosis with organ failure at admission called fulminant acute pancreatitis because of organ failure either at admission or within 72 hours (mortality 90 percent). [67]

Treatment of acute pancreatitis is aimed at correcting any underlying predisposing factors and at the pancreatic inflammation itself.



**Supportive Care**

The first step in managing patients with acute pancreatitis is determining whether the pancreatitis is likely to be mild or severe.

Mild acute pancreatitis is treated with supportive care including pain control, intravenous fluids, and correction of electrolyte and metabolic abnormalities. The majority of patients require no further therapy, and recover and eat within three to seven days.

In severe acute pancreatitis, intensive care unit monitoring and support of pulmonary, renal, circulatory, and hepatobiliary function may minimize systemic sequelae. [68]

Vital signs and urine output should be monitored every few hours in the first 24-48 hours. Patients with severe pancreatitis will need ongoing monitoring for other complications that might arise.

Fluid replacement is important because patients with necrotizing pancreatitis accumulate vast amounts of fluid in the injured pancreatic bed. Inadequate hydration can lead to hypotension and acute tubular necrosis. In addition, fluid depletion damages pancreatic microcirculation and results in pancreatic necrosis. At least one report suggested that inadequate fluid replacement (as evidenced by persistent

hemoconcentration at 24 hours) was associated with development of necrotizing pancreatitis. [69]

The exact amount and composition of fluid resuscitation that is required has not been extensively studied but several approaches have been published[70]. Adequate fluid replacement can be assessed by improvement of vital signs and urine output and reduction in hematocrit and blood urea nitrogen over 24 hours, particularly if they were high at the onset. Monitoring the blood urea nitrogen may be particularly important, as both the BUN at the time of admission and the change in BUN during the first 24 hours of hospitalization predict mortality. Increased fluid resuscitation should be considered in patients whose BUN levels stay the same or increases.

Fluid should be titrated to maintain urine output of greater than 0.5 cc/kg/hour. [71] A low urine output may already reflect the development of acute tubular necrosis rather than persistent volume depletion. In this setting, aggressive fluid replacement can lead to peripheral and pulmonary edema without improving the urine output. There is some evidence that fluid resuscitation with lactated Ringer's solution may be superior to normal saline [72].

Oxygen saturation needs to be assessed routinely and supplemental oxygen administrated to maintain arterial oxygen saturation of greater than 95 percent.

Deep vein thrombosis prophylaxis should be considered in bedridden patients.

**Pain Management**

Abdominal pain is often the dominant symptom. Uncontrolled pain can contribute to the hemodynamic instability. Adequate pain control is mandatory. Meperidine has been favored over morphine for analgesia in pancreatitis because studies showed that morphine caused an increase in sphincter of Oddipressure[73] . Fentanyl (intravenous) is being used increasingly for pain relief for all cases of mild AP due to its better safety profile, especially in renal impairment.

**Nutrition**

Patients with mild pancreatitis can often be managed with intravenous hydration alone since recovery often occurs rapidly, allowing patients to resume an oral diet. Nutritional support is often required in patients with severe pancreatitis.

Nutritional support should be provided to those likely to remain fasting for more than seven days. Nasojejunal tube feeding (using an

elemental or semi-elemental formula) is preferred to total parenteral nutrition.

**Enteral**

A benefit of early enteral nutrition is its ability to maintain the intestinal barrier. Bacterial translocation from the gut is probably a major cause of infection.

In a meta-analysis of eight trials, enteral nutrition significantly reduced mortality, multiple organ failure, systemic infections, and the need for surgery compared with those who received parenteral nutrition[74]. Guidelines issued by the American College of Gastroenterology and the American Gastroenterological Association recommended enteral feeding for severe acute pancreatitis .

Radiologic or endoscopic placement of a jejunal feeding tube beyond the ligament of Treitz and enteral feeding should be attempted. If not possible, nasogastric feeding has been proposed as an easier alternative. A controlled trial comparing nasogastric with nasojejunal feedings found no significant differences in any of the clinical outcomes measured [75].

The presence of fluid collections or elevated pancreatic enzymes is not necessarily a contraindication to oral or enteral feeding. However, in

a subgroup of patients there is clear correlation of pain, recurrence of pancreatitis, or worsening of fluid collections to feeding, either oral or enteral. These patients often have disrupted pancreatic ducts with fluid collections. Drainage of fluid collections may allow resumption of oral intake. If the fluid collections are not considered suitable for drainage, total parenteral nutrition will be needed to maintain nutrition. If the target rate of enteral feeding is not achieved within 48 to 72 hours, supplemental parenteral nutrition should be provided.

**Parenteral**

Parenteral nutrition should be initiated in patients who do not tolerate enteral feeding or in whom nutritional goals cannot be reached within two days.

**Control of Infection**

The occurrence of pancreatic infection is a leading cause of morbidity and mortality in acute necrotizing pancreatitis. Approximately one-third of patients with pancreatic necrosis develop infected necrosis[11]. Patients who develop infection tend to have more extensive necrosis. Although infection can occur early in the course of necrotizing pancreatitis, it is more often seen late in the clinical course (after 10 days) [76].

The important organisms causing infection in necrotizing pancreatitis are predominantly gut-derived, including Escherichia coli, Pseudomonas, Klebsiella, and Enterococcus. The majority of infections (about 75 percent) are monomicrobial. Fungal infection and infection with gram-positive organisms are uncommon but occur more frequently in the setting of prophylactic antibiotic use for severe acute pancreatitis, especially when used for more than 10 to 14 days. Fungal infections occur in approximately 9 percent of necrotizing pancreatitis and it is not clear if they are associated with higher mortality.

Approaches taken to decrease bacterial infections in acute necrotizing pancreatitis include enteral feeding, systemic antibiotics, percutaneous computerized tomography (CT) guided aspiration, and necrosectomy.

**Role of Systemic antibiotics**

The role of prophylactic systemic antibiotics in acute pancreatitis is unsettled since studies evaluating its benefits and harms[77] have produced contradictory results. Guidelines have been issued by multiple societies and differ in their recommendations

The American College of Gastroenterology guidelines do not recommend prophylactic antibiotics.

Guidelines from the American Gastroenterological Association[153] do not make a firm recommendation with regard to prophylactic antibiotics, but note that "Antibiotic prophylaxis, if used, should be restricted to patients with substantial pancreatic necrosis (>30 percent of the gland necrotic by CT criteria) and should continue for no more than 14 days."

Guidelines from the Italian Association for the Study of the Pancreas[79] recommend them for patients with CT-proven necrosis.

**Protease inhibitors**

Protease inhibitors, a class of drugs used to treat or prevent infection by viruses, have been described in the treatment of acute pancreatitis in observational studies, but their role remains unclear [80].

**Percutaneous CT-guided aspiration**

CT-guided percutaneous aspiration with gram's stain and culture is recommended when infected pancreatic necrosis is suspected. Sterile necrosis does not usually require antibiotics and acute fluid collections do not require therapy in the absence of infection or obstruction of surrounding hollow viscous.

## Necrosectomy

Surgical debridement of infected necrosis (necrosectomy) can be accomplished by open surgery or a minimally invasive approach (endoscopic or percutaneous radiologic). Indications for necrosectomy include a failure to improve after antibiotics and CT-guided aspiration or if the patient becomes unstable from pulmonary, cardiovascular, or renal complications.

Because of the high mortality and morbidity associated with early necrosectomy, patients may benefit from continued conservative management. [81,82]

## Treatment of Associated Conditions

In addition to the above treatment for pancreatic inflammation, treatment of acute pancreatitis is aimed at correcting any underlying predisposing factors, such as gallstones, hypertriglyceridemia, and complications of splenic vein thrombosis.

## Gallstone Pancreatitis

Gallstone pancreatitis requires specific therapeutics considerations. In this disorder, obstructive stones in the biliary tract or ampulla of Vater are responsible for the pancreatitis.

**Endoscopic retrograde cholangiography**

Early endoscopic retrograde cholangiography (ERCP) with papillotomy or surgical intervention to remove bile duct stones may lessen the severity of gallstone pancreatitis. Multiple studies suggest that early endoscopic papillotomy is of benefit in humans with acute biliary pancreatitis.

ERCP should be performed within 24 hours if there is concomitant cholangitis. In general, ERCP should be performed within 72 hours in those with a high suspicion of persistent bile duct stones (ie, visible common bile duct stone on noninvasive imaging, persistently dilated common bile duct, and jaundice).

Early ERCP in those with predicted or actual severe gallstone pancreatitis in the absence of cholangitis or a high suspicion of a persistent common bile duct stone is controversial[83,84].

**Cholecystectomy**

Cholecystectomy should be performed after recovery in all patients with gallstone pancreatitis prior to hospital discharge. It is indicated only after an attack of acute pancreatitis since the incidence of pancreatitis from gallstones is only 3 to 7 percent. Failure to perform cholecystectomy is associated with a 25 to 30 percent risk of recurrent acute pancreatitis, cholecystitis, or cholangitis within 6 to 18 weeks. The risk is highest in patients who did not undergo asphincterotomy.

In patients who have had mild pancreatitis, cholecystectomy can usually be performed safely within seven days after recovery. On the

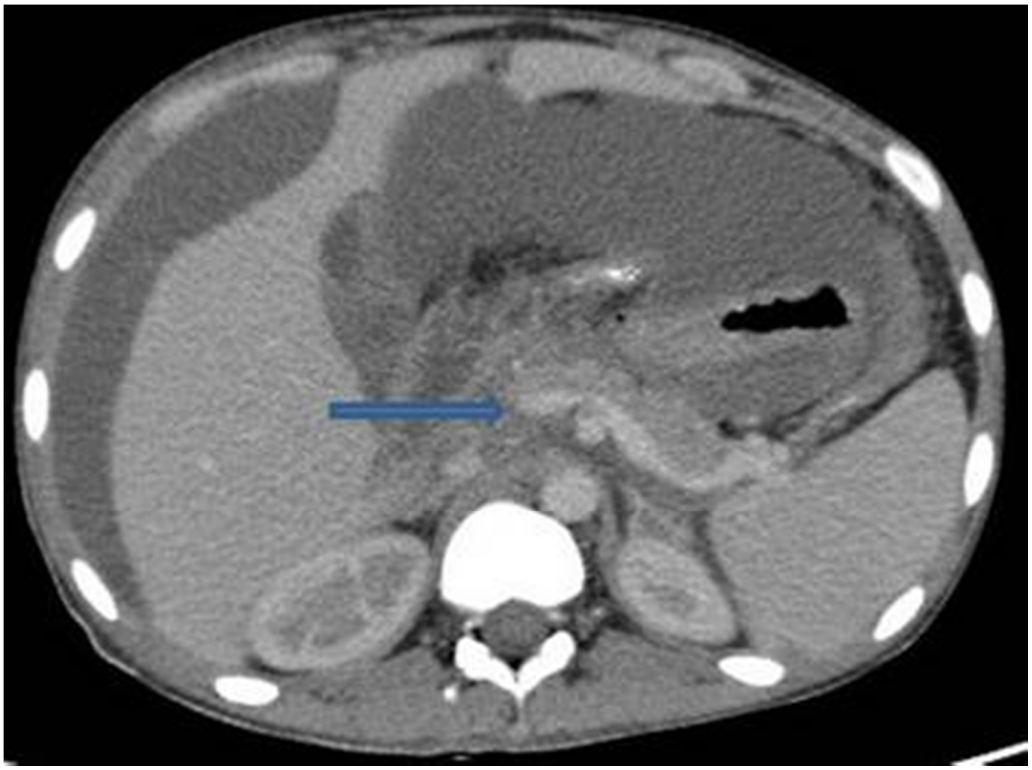
other hand, in patients who have had severe necrotizing pancreatitis, delaying cholecystectomy for at least three weeks may be reasonable because of an increased risk of infection. There is some controversy about cholecystectomy after sphincterotomy in elderly patients.

Pancreatitis occurring in patients with gallstones suggests that there has been migration of stones into the common bile duct. Thus, a cholangiogram and clearance of the common bile duct if stones present either before or during surgery is mandatory to prevent recurrence after cholecystectomy. If the clinical suspicion of common bile duct stones is high (eg, in those with persistent or worsening liver test abnormalities or cholangitis), a preoperative ERCP is the best test as there is a high likelihood that therapeutic intervention (sphincterotomy, stone extraction) will be required. On the other hand, if the suspicion of persistent common bile duct stone is low (eg, if liver test normalize), an intraoperative cholangiogram during cholecystectomy may be preferable to avoid the morbidity associated with ERCP.

Magnetic resonance cholangiopancreaticography and endoscopic ultrasound are other imaging options that can exclude common bile duct stones[85]. A preoperative ERCP can then be performed only in those with stones or sludge in the common bile duct.

**Splenic Vein Thrombosis**

Splenic vein thrombosis is seen in up to 19 percent of patients[86]. Treatment should focus on the underlying pancreatitis since the effective treatment may be associated with spontaneous resolution of the thrombosis. Anticoagulation may be needed if there is extension of the clot into the portal or superior mesenteric vein resulting in hepatic decompensation or compromise of bowel perfusion. However, this needs to be considered along with the theoretical possibility of hemorrhage into pancreatic necrosis or fluid collections.

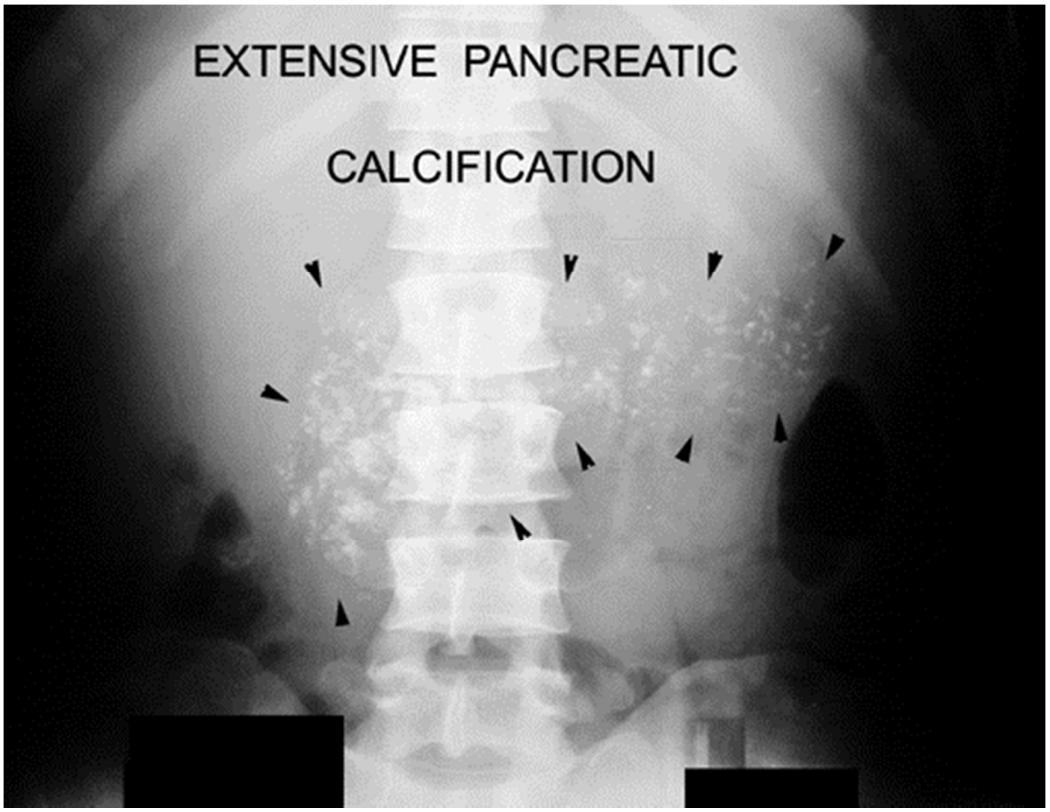


**Abdominal compartment syndrome**

Patients with severe pancreatitis are at increased risk for intraabdominal hypertension and abdominal compartment syndrome. Factors that can contribute to abdominal compartment syndrome in patients with acute pancreatitis include tissue edema from aggressive fluid resuscitation, peripancreatic inflammation, ascites, and ileus[87]. If abdominal compartment syndrome is confirmed, either percutaneous catheter-based or surgical decompression is indicated .

**Chronic pancreatitis**

Chronic pancreatitis is inflammation of the pancreas that does not heal or improve—it gets worse over time and leads to permanent damage. Chronic pancreatitis eventually impairs a patient's ability to digest food and make pancreatic hormones




**Pain Management**

Significant pain associated with chronic pancreatitis can seriously reduce a patient's quality of life. It is important to treat chronic pancreatitis as soon as it is diagnosed because repeated episodes of inflammation can cause irreversible damage, and pain relief becomes much less effective. Pain relief can be achieved with medication, often using the World Health Organization's 3-step ladder approach to pain relief:

1. Pain medication begins with nonopioids (like acetaminophen, ibuprofen, or both).

2. If nonopioids do not relieve pain, mild opioids (like codeine) are given.

3. If mild opioids do not relieve pain, strong opioids (like morphine) are given.

Many patients with chronic pancreatitis receive antioxidants with their pain medicine, which has been shown to help with pain relief.3-5 There are other options for pain relief, such as a celiac plexus block, which may provide another option for significant pain relief. The celiac plexus block is achieved via injection and prevents the nerves that travel from the pancreas from reporting pain signals back to the brain.

If there is a narrowing of the pancreatic duct, placement of a plastic tube called a stent into the duct can be helpful in alleviating pain symptoms.

**Limited Role of Endoscopic Retrograde Cholangiopancreatography (ERCP)**

An ERCP test, in which a flexible endoscope is placed into the intestine and a catheter is used to inject dye into the pancreas, should

generally not be used in chronic pancreatitis, and it should never be used to diagnose chronic pancreatitis because injecting dye into the pancreas can cause pancreatitis.

**Surgery**

When medical therapy fails to provide relief to patients with chronic pancreatitis, surgical therapy may be an option. A lateral pancreaticojejunostomy (modified Puestow procedure) can result in pain relief in up to 80% of patients.

**Whipple Procedure**

Another surgical procedure, which can remove inflammation and masses on the head of the pancreas, is the classic Whipple procedure; however, this procedure does remove a lot of important tissue and can be associated with complications such as increased risk of death. When possible, modified Whipple procedures are performed to save more tissue compared to the classic Whipple procedure, and can be successful for pain relief and return to daily activity. To read more,

## TPIAT

For appropriately selected patients whose pain remains incapacitating despite standard medical and surgical approaches, total pancreatectomy with islet auto-transplantation (TP-IAT) – while not a panacea – yields significant relief of symptoms.

## Antioxidant therapies

Basic and clinical evidence suggests that the development of both acute pancreatitis (AP) and chronic pancreatitis (CP) can be associated with oxidative stress. Findings show that free radical activity and oxidative stress indices are higher in the blood and duodenal juice of patients with pancreatitis.

Based on these findings, the idea of using antioxidant regimens in the management of both AP and CP as a supplement and complementary in combination with its traditional therapy is reasonable. In practice, however, the overall effectiveness of antioxidants is not known, and the best mixture of agents and dosages is not clear. Currently, a trial of a mixture of antioxidants containing vitamin C, vitamin E, selenium, and methionine is reasonable as one component of overall medical management.

In summation, there is no definite consensus on the dosage, length of therapy, and ultimately, the benefits of antioxidant therapy in the management of AP or CP. Further well-designed clinical studies are needed to determine the appropriate combination of agents, time of initiation, and duration of therapy.

# MATERIALS AND METHODS

**DESIGN OF STUDY:** Randomized prospective comparative clinical study.

Minimum of 100 consecutive patient diagnosed with acute pancreatitis of 72 hrs or less duration

**INCLUSION CRITERIA:**

Patients diagnosed with acute pancreatitis based on the following 2 criteria:

1. Abdominal pain characteristic of acute pancreatitis (duration <72 hrs)
2. Serum amylase and/or lipase ≥ 3times the upper limit of normal

**EXCLUSION CRITERIA: Patients**

1. Sensitive to LMWH
2. Pregnant
3. Breast feeding
4. Coagulation disorders
5. Undergoing haemodialysis

**SOURCE OF DATA**

patients admitted in COIMBATORE MEDICAL COLLEGE from june 2016 to august 2017

**METHODS OF DATA COLLECTION**

Patients who satisfy the inclusion criteria will be enrolled into the study. Prior informed consent will be taken from the patients and relatives before enrolling them into the study. The particulars of the patient including name, age, gender, admitting diagnosis will be documented.

Patients will be assigned to 2 groups as random number table A and B.

**GROUP A patients**

will undergo conventional therapy that includes management of shock ,maintenance of water and electrolytes balance, fasting, gastrointestinal decompression , administration of pancreatic enzymes inhibitor(octreotide) , antibiotics (cephalosporins and metronidazole) and oral manganese sulfate and symptomatic treatment.

**GROUP B patients:**

The treatment included following the methods used in GROUP A patients, plus administering LMWH at 100 micro gram /kg per day

subcutaneous injection starting from the admission day and continuing for 7 days.

## OBSERVATION PARAMETERS

**Clinical parameters:** Clinical Severity by Atlanta Criteria(Complication rate, occurrence rate of organ failure), in hospital mortality, curative rate and mean hospital stay in the two groups were observed and compared.

**Laboratory tests:**

The APACHE II scores, parameters on admission and at 1 week after treatment were determined:

## STATISTICAL METHODS

Parametric and non parametric data will be analyzed using students t-test (paired and unpaired) and Chi- square test respectively. A P value<5 will be considered significant.

# RESULTS

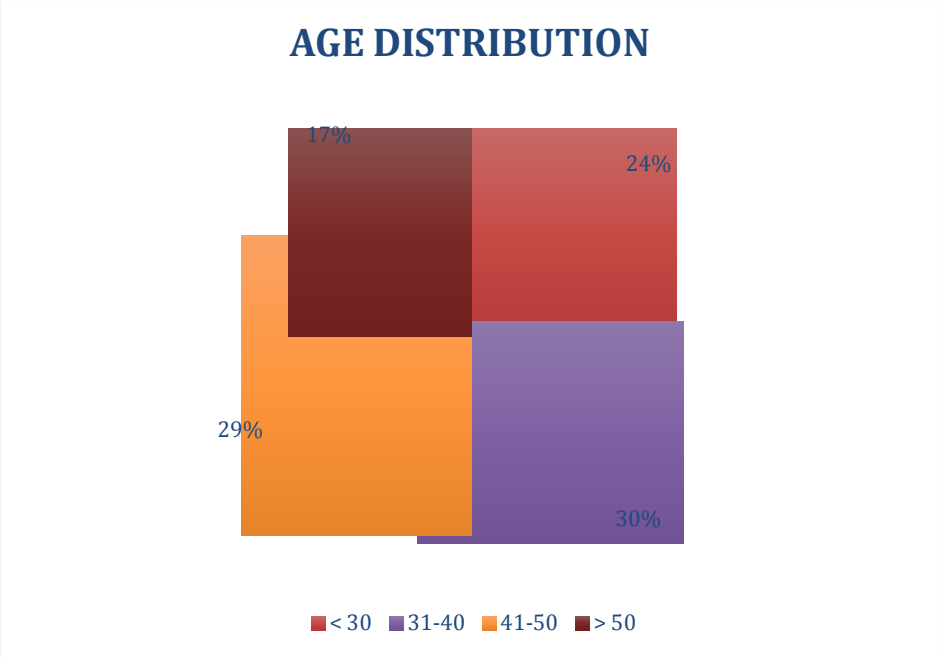
## 1. **Comparison between groups**

In our study, there were 50 patients in the observation group (44 men, 6 women; average age 40 years) and 50 patients in the control group (42 men, 8 women; average age 40.04 years). Differences in general data, such as sex and age, between the groups were not statistically significant ($p > 0.05$). Thus, the groups were comparable.

## AGE DISTRIBUTION

## TABLE-1

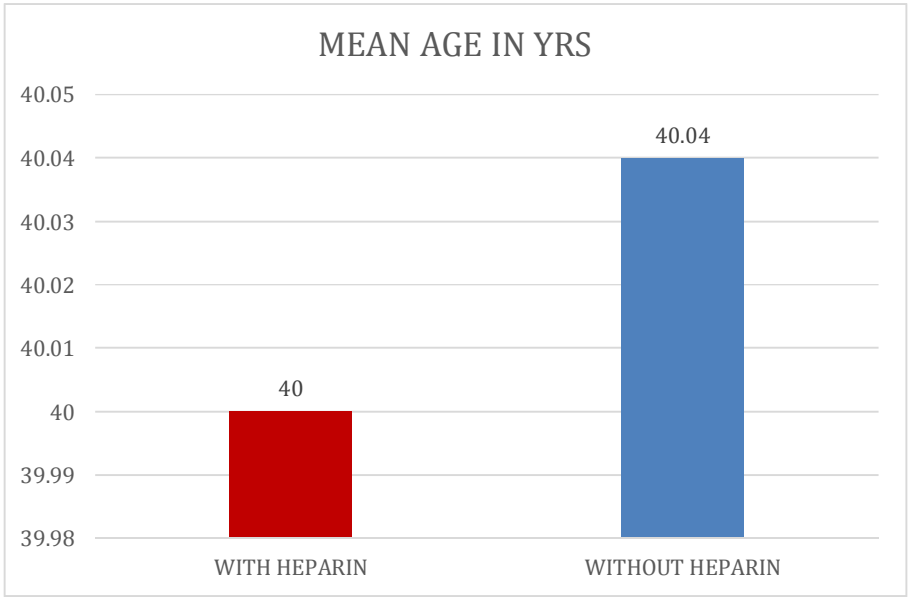
| AGE(IN YRS) | NO OF PATIENTS | PERCENTAGE |
|---|---|---|
| < 30 | 24 | 24% |
| 31-40 | 30 | 30% |
| 41-50 | 29 | 29% |
| > 50 | 17 | 17% |



## AGE IN YEARS

## TABLE-2

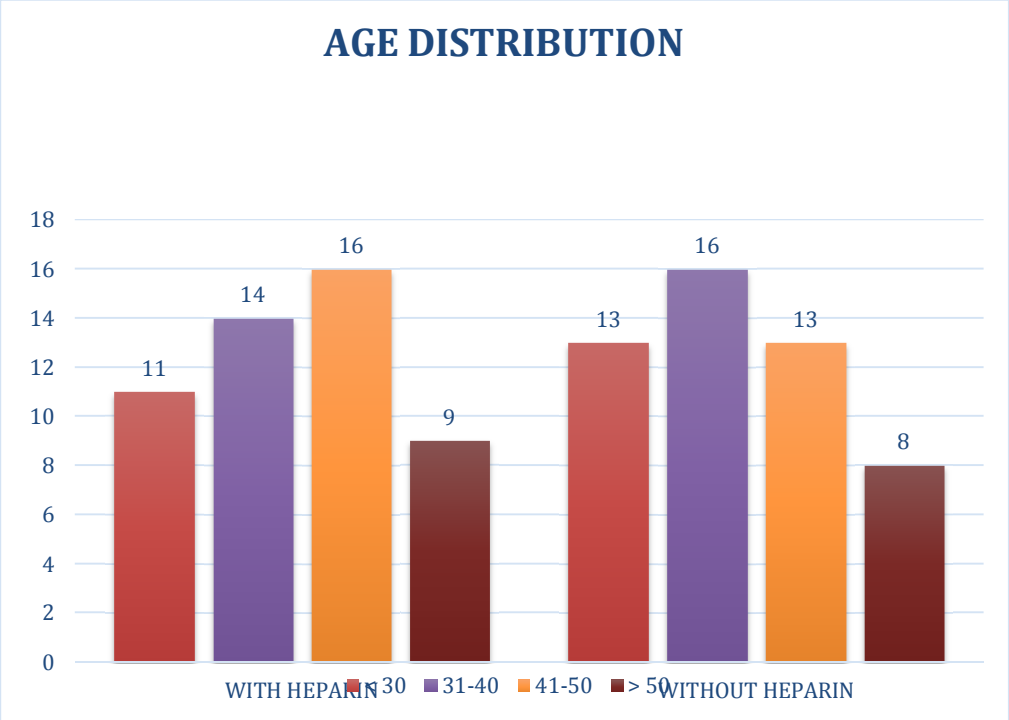
| | AGE IN YEARS | |
|---|---|---|
| TREATMENT | MEAN | SD |
| WITH HEPARIN | 40 | 9.44 |
| WITHOUT HEPARIN | 40.04 | 11.92 |
| | | |
| P VALUE - 0.985 | | |
| NON SIGNIFICANT | | |
| UNPAIRED T TEST | | |



## AGE DISTRIBUTION AMONG GROUPS

### TABLE-3

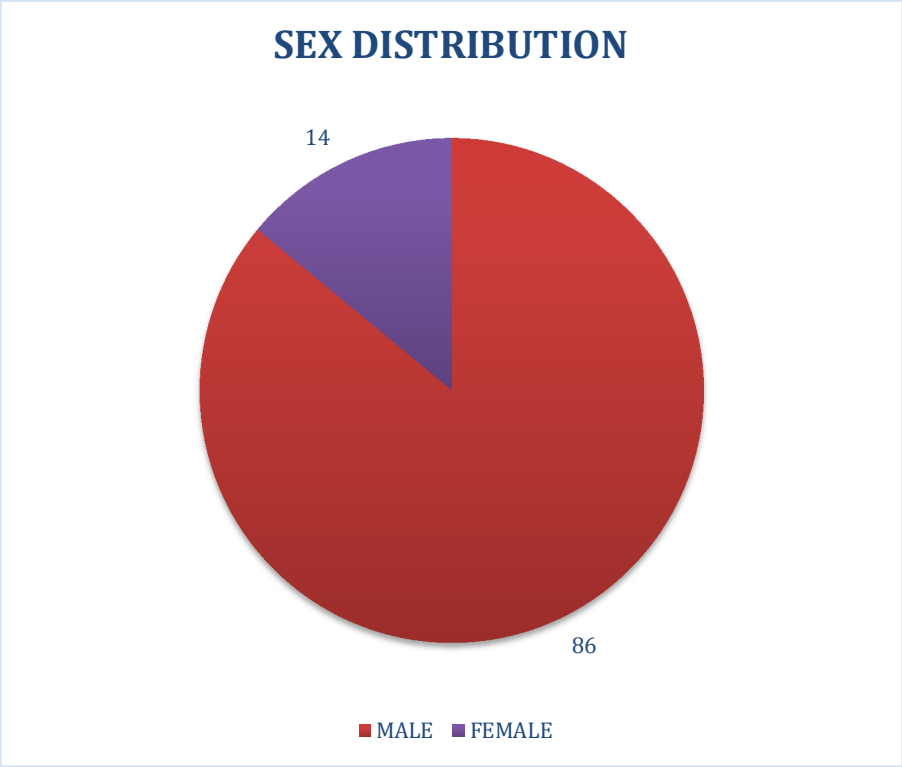
| | TREATMENT | |
|---|---|---|
| AGE(IN YRS) | WITH HEPARIN | WITHOUT HEPARIN |
| < 30 | 11 | 13 |
| 31-40 | 14 | 16 |
| 41-50 | 16 | 13 |
| > 50 | 9 | 8 |
| | | |
| P VALUE - 0.880 | | |
| NON SIGNIFICANT | | |
| KRUSKAL WALLIS TEST | | |



## SEX DISTRIBUTION

## TABLE-4

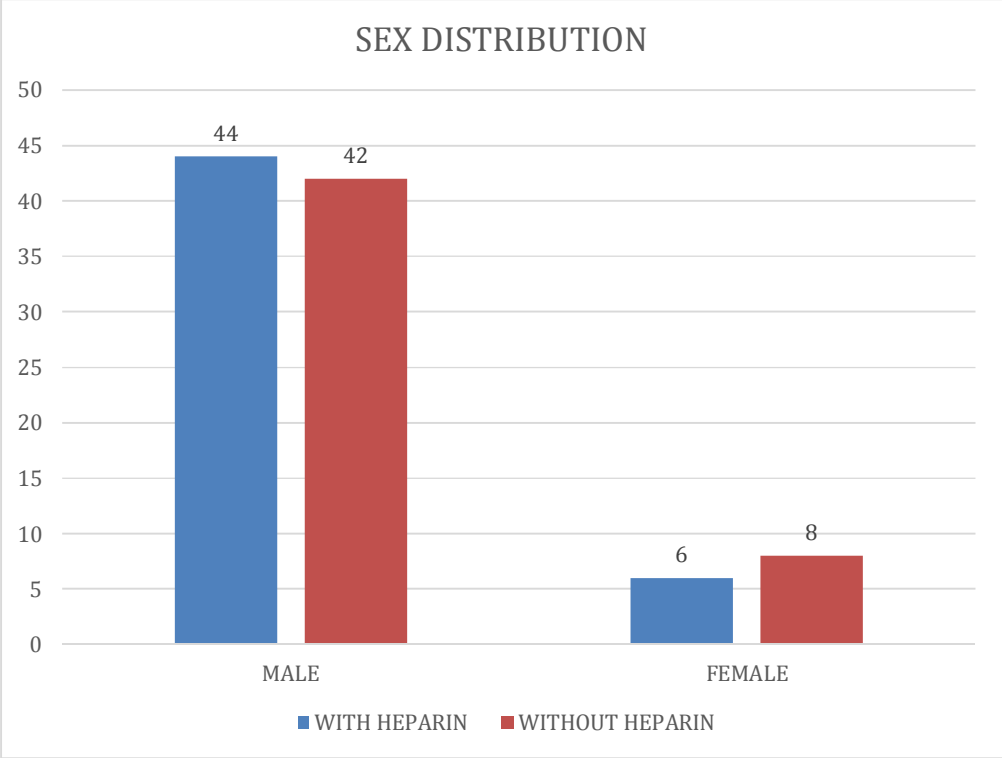
| SEX | NO OF PATIENTS | PERCENTAGE |
|---|---|---|
| MALE | 86 | 86% |
| FEMALE | 14 | 14% |



## SEX DISTRIBUTION AMONG GROUPS

## TABLE-5

| | TREATMENT | |
|---|---|---|
| SEX | WITH HEPARIN | WITHOUT HEPARIN |
| MALE | 44 | 42 |
| FEMALE | 6 | 8 |
| | | |
| P VALUE - 0.564 | | |
| NON SIGNIFICANT | | |
| MANN WHITNEY U TEST | | |



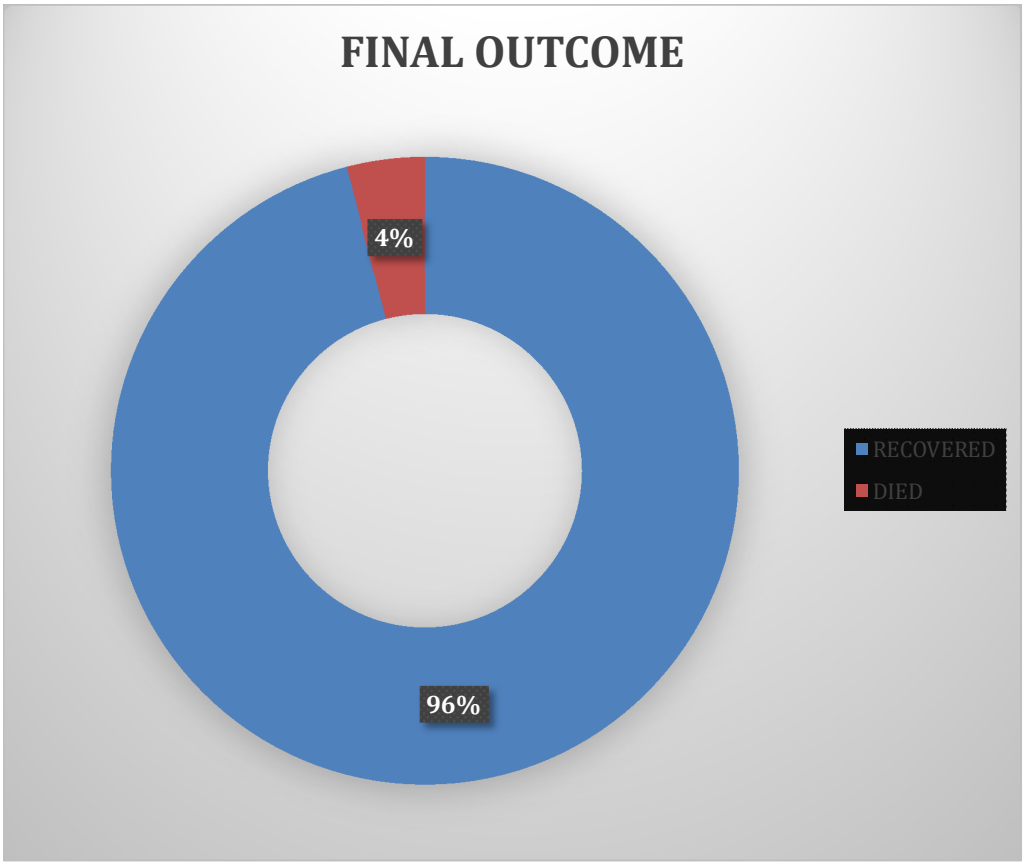
## 2. Changes in final outcome after treatment

In our study out of 100 patients 96 patients recovered and 4 patients died. All the 4 patients were from control group. The final outcome of recovery rate between the two grops were statistically significant with ($p < 0.05$), (Table 7)

**FINAL OUTCOME**

**TABLE-6**

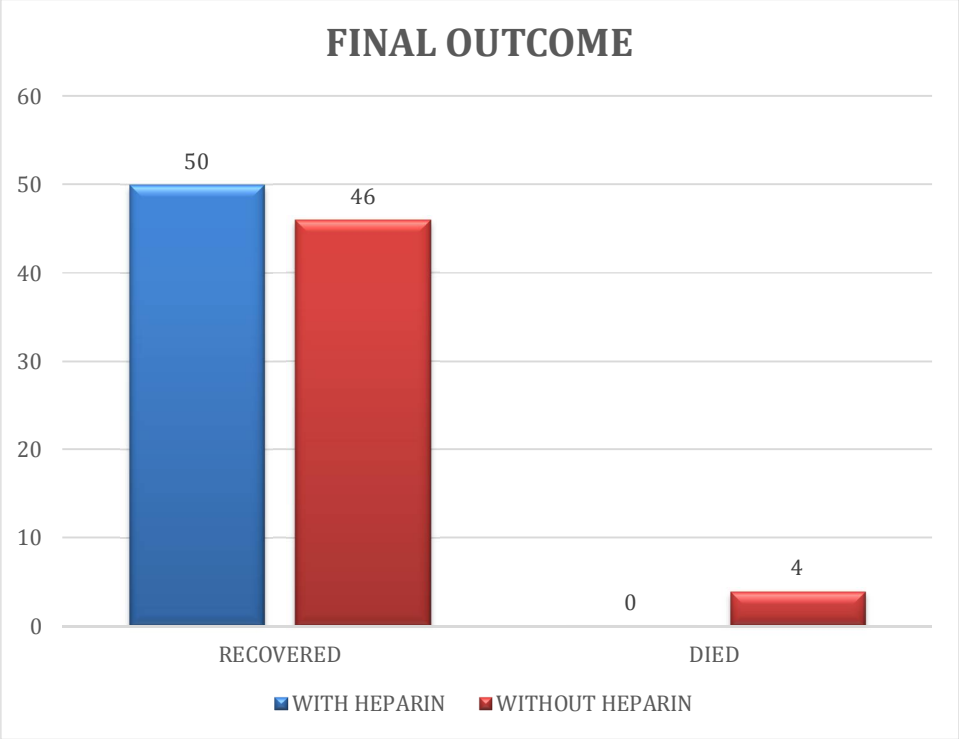
| FINAL OUTCOME | NO OF PATIENTS | PERCENTAGE |
|---|---|---|
| RECOVERED | 96 | 96% |
| DIED | 4 | 4% |


FINAL OUTCOME
4%
96%
RECOVERED
DIED

## FINAL OUTCOME AMONG GROUPS

## TABLE-7

| | TREATMENT | |
|---|---|---|
| FINAL OUTCOME | WITH HEPARIN | WITHOUT HEPARIN |
| RECOVERED | 50 | 46 |
| DIED | 0 | 4 |
| | | |
| P VALUE - 0.041 | | |
| SIGNIFICANT | | |
| CHI SQUARE TEST | | |



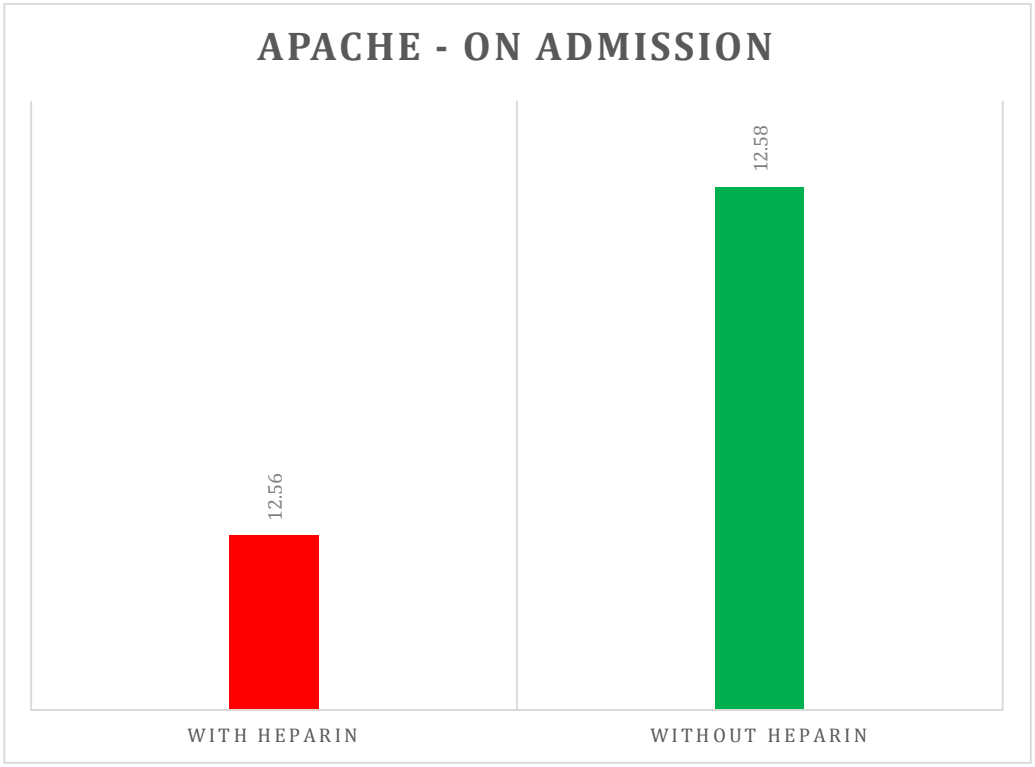
3. **Changes in APACHE scores after treatment**

The APACHE II scores at admission were not significantly different between the groups ($p > 0.05$), (Table 8). After 7 days of treatment, the APACHE II scores in the observation group (i.e. patients treated with heparin) were significantly lower than those in the control group with ($p < 0.05$), (Table 9).

**TABLE-8**

**APACHE SCORE - ON ADMISSION**

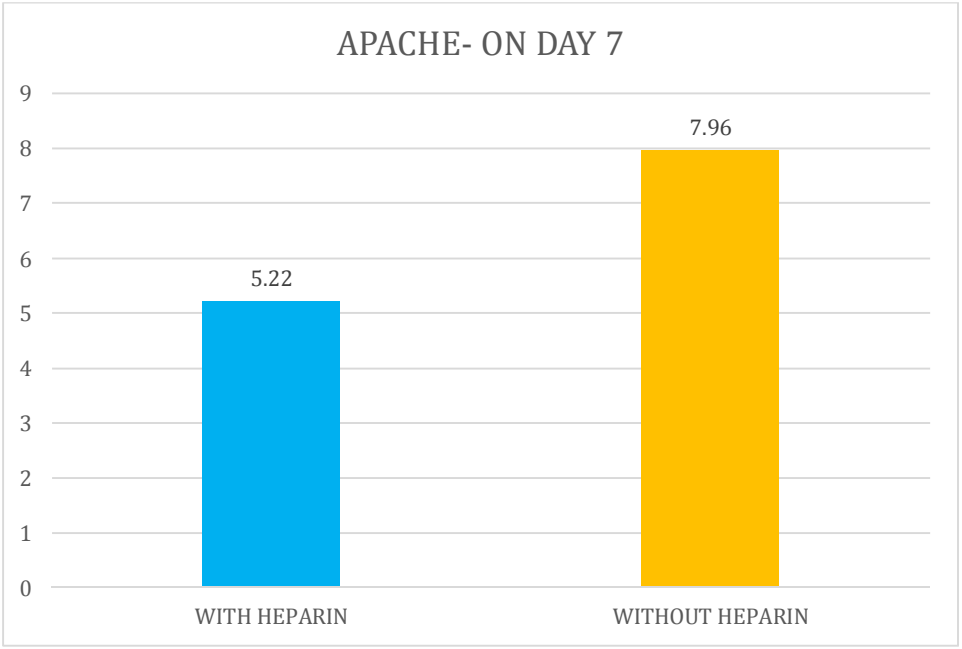
| | APACHE SCORE - ON ADMISSION | |
|---|---|---|
| TREATMENT | MEAN | SD |
| WITH HEPARIN | 12.56 | 5.85 |
| WITHOUT HEPARIN | 12.58 | 6.83 |
| | | |
| P VALUE - 0.987 | | |
| NON SIGNIFICANT | | |
| UNPAIRED T TEST | | |


APACHE - ON ADMISSION
12.56
12.58
WITH HEPARIN
WITHOUT HEPARIN

## APACHE SCORE- ON DAY 7

## TABLE-9

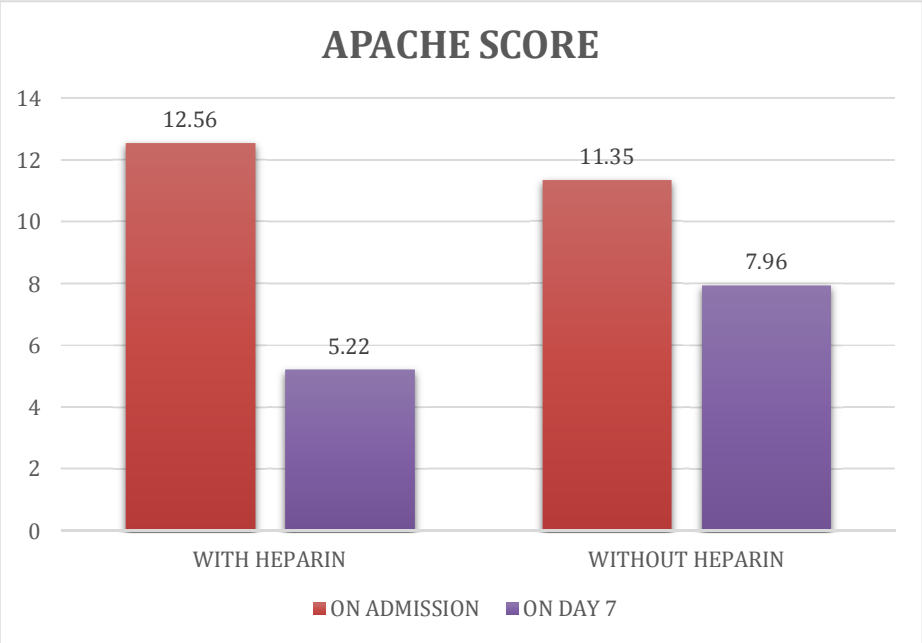
| | APACHE SCORE- ON DAY 7 | |
|---|---|---|
| TREATMENT | MEAN | SD |
| WITH HEPARIN | 5.22 | 3.79 |
| WITHOUT HEPARIN | 7.96 | 4.65 |
| | | |
| P VALUE - 0.002 | | |
| SIGNIFICANT | | |
| UNPAIRED T TEST | | |



## TREATMENT

## TABLE-10

| | TREATMENT | |
|---|---|---|
| APACHE SCORE | WITH HEPARIN | WITHOUT HEPARIN |
| ON ADMISSION | 12.56 | 11.35 |
| ON DAY 7 | 5.22 | 7.96 |
| **MEAN DIFFERENCE** | **7.34** | **3.39** |
| P VALUE -0.005 | | |
| SIGNIFICANT | | |

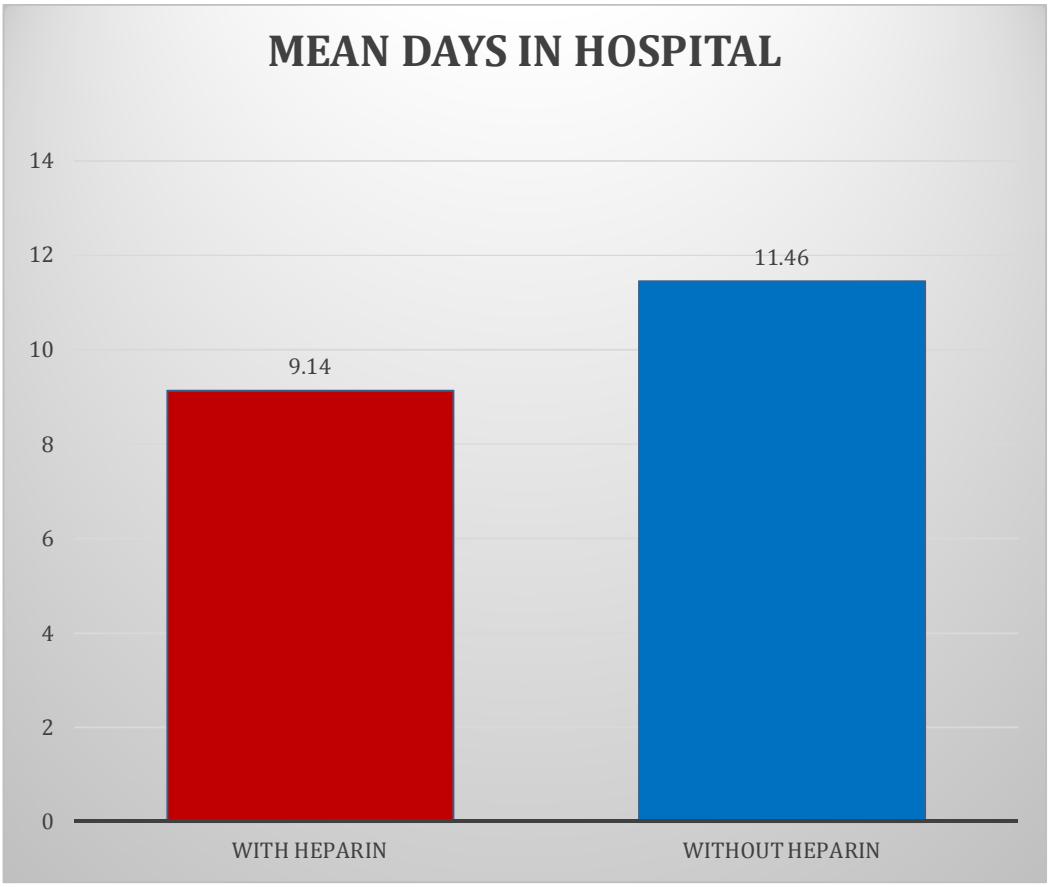


**4. Changes in duration of hospital stay**

In our study mean duration of hospital stay in patients treated with heparin was 9.14 days and in patients treated without heparin was 11.46 days. Which were statistically significant between the two groups with ($p<0.05$), (Table 11).

**TABLE-11**

| | DAYS IN HOSPITAL | |
|---|---|---|
| TREATMENT | MEAN | SD |
| WITH HEPARIN | 9.14 | 2.68 |
| WITHOUT HEPARIN | 11.46 | 5.41 |
| | | |
| P VALUE - 0.008 | | |
| SIGNIFICANT | | |
| UNPAIRED T TEST | | |


MEAN DAYS IN HOSPITAL
14
12
10
8
6
4
2
0
9.14
11.46
WITH HEPARIN
WITHOUT HEPARIN

# DISCUSSION

Although research on the pathogenesis and mechanisms of severe acute pancreatitis has progressed in recent years, the primary cause of the high mortality remains unknown. In early stages of pancreatitis, macrophages, neutrophils, endothelial cells are activated. Preinflammatory cytokines are released and inflammation factors re elevated during acute pancreatitis and have been implicated in progression of pancreatitis associated microvascular disturbance and hemorrhagic necrosis. Ischemia, reperfusion injury and tiny thrombosis are closely associated with pancreatic microcirculation disturbance[1]

As an anticoagulation drug, LMWH can effectively restrain the activity of thrombin and blood coagulation factor Xa, inhibit platelet aggregation, and improve the microcirculation. LMWH can also reduce inflammation by lowering the expression of pro-inflammatory factors, inflammatory factors, adhesive factors.

In this study, the curative effects of adding lmwh alone in the treatment of acute pancreatitis were compared with conventional strategy. The results showed obvious improvements in laboratory indices, much higher cure rate, and lower incidence of complications in the observation than in the control group, suggesting that LMWH safe and effective for treatment of severe acute pancreatitis.

Furthermore, the results showed that the decline in APACHE-II scores in the observation group was larger than that in the control group and lower values than those in C group ($p < 0.05–0.01$). The results of our study is on par with other international studies[5,6,7]

APACHE-II scores show an obvious increase in the early stage (0 – 48 h) and the increase becomes faster at 24 to 48 h. A continued increase in APACHE-II scores after discharge usually indicates disease progression. In this study, the APACHE-II scores in the observation group were much lower than those in the control group after treatment, suggesting that LMWH can relieve acute pancreatitis-related inflammation and reduce the incidence of complications.

**Limitations of the study**

This study found that LMWH had a marked effect in the treatment of acute pancreatitis. However, our study is a small, population based study from a single institution, from among patients admitted during a brief time-frame. Larger, multi institutional studies with larger sample size and longer time frames are needed to conform validity and accuracy my results.

Patients were followed up till the time of discharge. Further follow up; to analyze further complications of acute pancreatitis did not fall under the parameters of our study.

Pediatric patients were not include in our study

# CONCLUSION

Findings From Our Study Found that use of LMWH in treatment of Acute pancreatitis, which acts by improving microcirculation is an effective drug in the non-surgical treatment of acute pancreatitis

As per our study, the APACHE II Scores reduced considerably in the group treated with LMWH, suggesting that there was a considerable improvement in laboratory values, higher cure rate and lower complication. Such as Necrosis, Abscess, sepsis and Organ failure etc.Thus LMWH can effectively relieve acute pancreatitis related inflammation and reduce incidence of complications

LMWH can rapidly relieve abdominal pain, halt the progression of diseases, reduce the severity and complication, shorten the length of hospital stay and enhance the cure rate

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# PROFORMA

PATIENT DATA COLLECTION FORM

Patient NO:

DEMOGRAPHIC DATA

Age: Sex:

## 1) APACHE-II

## The APACHE II Severity of Disease Classification System

| | HIGH ABNORMAL RANGE | | | | | LOW ABNORMAL RANGE | | | |
|---|---|---|---|---|---|---|---|---|---|
| **Physiological variable** | **+4** | **+3** | **+2** | **+1** | **0** | **+1** | **+2** | **+3** | **+4** |
| **TEMPERATURE** ( ° C ) | > 41° | $39^0$- 40.8 ° | | 35.5° - 38.9 ° | 36 ° - 38.4 ° | 34 ° - 35.8 ° | 32 ° - 33.9 ° | 30°- 31.9 ° | < 29.9 ° |
| **MEAN ARTERIAL PRESSURE** (mm Hg) | > 160 | 130 - 159 | 110-129 | | 70 - 109 | | 50-69 | | < 49 |
| **HEART RATE** (ventricular response) | > 180 | 140 - 178 | 110-139 | | 70 - 109 | | 55-69 | 40-54 | < 39 |
| **RESPIRATORY RATE** (non ventilated or ventilated) | > 50 | 35 - 49 | | 25-34 | 12 - 24 | 10-11 | 6 - 9 | | < 5 |
| **OXYGENATION** | > 500 | 350 - 498 | 200 - 349 | | < 200 PO, > 70 | 200 - 349 PO, 61 - 70 | 200 - 349 | 200 - 349 PO, 55-60 | 200 - 349 PO, < 55 |
| **ARTERIAL** pH | > 7.7 | 7.6 - 7.69 | | 7.5 - 7.59 | 7.33 - 7.49 | | 7.15 - 7.32 | 7.15 - 7.24 | < 7.15 |

| | HIGH ABNORMAL RANGE | | | | | LOW ABNORMAL RANGE | | | |
|---|---|---|---|---|---|---|---|---|---|
| **Physiological variable** | **+4** | **+3** | **+2** | **+1** | **0** | **+1** | **+2** | **+3** | **+4** |
| | | | | | | | | | |
| **SERUM SODIUM** (meg/dl) | > 180 | 160 - 179 | 155 - 159 | 150- 154 | 130 - 149 | | 120 - 129 | 111 - 119 | < 110 |
| **SERUM POTASSIUM** (meg/dl) | > 7 | 6 - 6.9 | | 5.5 - 5.9 | 3.5 - 5.4 | 3 - 3.4 | 2.5 - 2.9 | | < 2.5 |
| **SERUM CREATININE** (mg/100 ml) | > 3.5 | 2 - 3.4 | 1.3 - 1.9 | 0.6 - 1.4 | | | < 0.6 | | |
| **HEMATOCRIT** (%) | > 60 | | 50 - 59.9 | 46 - 49.9 | 30 - 45.9 | | 20-29.9 | | < 20 |
| **WHITE BLOOD COUNT** (total/mm3) (in 1000s) | > 40 | | 20 - 39.9 | 15 - 19.9 | 3 - 14.9 | | 1-2.9 | | < 1 |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| **GLASGOW COMA SCORE** (GCS) (score 15 minus actual GCS) | | | | | | | | | |
| **Total ACUTE PHYSIOLOGY SCORE**(APS) Sum of the 12 individual variable points | | | | | | | | | |
| SERUM HCO3 | > 52 | 41 - 51.9 | | 32 - 40.9 | 22 - 31.9 | | 18 - 21.9 | 15 - 17.9 | < 15 |

**AGE POINTS**

**Assign points to age as follows:**

| AGE (yrs) | POINTS |
|---|---|
| < 44 | 0 |
| 45 - 54 | 2 |
| 55 - 64 | 3 |
| 65 - 74 | 5 |
| > 75 | 6 |

**CHRONIC HEALTH POINTS**

If the patient has a history of severe organ system

a. For nonoperative or emergency postoperative patients (5 points)

b. for selective postoperative patients (2 points)

**DEFINITIONS:**

Organ insufficiency or immuno-compromised state must have been evident prior to the hospital admission and conform to the following criteria.

**LIVER:**

Biopsy proven cirrhosis and documented portal hypertension episodes

**RESPIRATORY:**

Chronic restrictive, obstructive, or vascular disease resulting in severe exercise restriction.

Unable to climb stairs or perform household duties: or documented chronic hypoxia, hypercapnia, secondary polycythemia, severe pulmonary hypertension ( >

| | | |
|---|---|---|
| deficiency or is immuno - compromised, assign points as follows: | of past upper GI bleeding attributed to portal hypertension: prior episodes of hepatic failure/encephalopathy /coma. | 40mmHg), or respirator dependency.<br><br>**RENAL:** Recurring chronic dialysis<br><br>IMMUNO-COMPROMISED: The patient has received therapy that suppresses resistance to infection [e.g. immuno-suppression, chemotherapy, radiation, long-term or recent high dose steroids] or has a disease that is |

| | | |
|---|---|---|
| | | sufficiently advanced to suppress resistance to infection [e.g. leukemia, lymphoma, AIDS] |

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**APACHE SCORE [ sum of A+B+C ]**

**A. APS points**

**B. Age points**

**C. Chronic health points**

**TOTAL APACHE SCORE**

# CONSENT FORM

PART 1 0F 2

Dear volunteers,

We welcome you and thank you for your interest in participation in this research project. Before you participate in this study , it is important for you to understand why this research is beign carried out.this form wil explain you all the relevant details of this research . it will explain the nature,the purpose,the benefits,the risks, the discomforts,the precautions and the information about how this project will be carried out. It is important that you read and understand the contentsof the form carefully.

**TITLE OF THE PROJECT**: "THE ROLE OF LOW MOLECULAR WEIGHT HEPARIN IN TREATMENT OF ACUTE PANCREATITIS

Name of the investigator:

**What is the purpose of this project/study?**

To establish the use of low molecular weight heparin in treatment of acute pancreatitis

**How the study will be carried out?**

All patients of acute pancreatitis who the fit inclusion criteria will be included in study. For this research only data will be collected and no intervention will be done.

- Following datas will be collected:

  Demographics (age,gender)
- Etiology
- Blood investigations
    a) Complete blood count
    b) Blood urea and creatinine
    c) Serum electrolytes
    d) Vitals monitoring
- Clinical and severity scores

Following is the Inclusion criteria: All patients admitted with the diagnosis of Acute Pancreatitis based on the presence of at least two of the following three criteria:

1. Characteristic epigastric abdominal pain , with or without radiation to the back.
2. Serum amylase or lipase levels elevated to at least three times the upper limit of normal.

3. Characteristic finding of Acute Pancreatitis on abdominal CT scan.

**What is the expected duration of the subject participation?**

No additional stay in hospital is required than the usual course.

**What are the benefits to be expected from the research to the participant or to others and the post-trial responsibilities of the investigator?**

At the end of study we will be able to find the use of LMW Heparin in treatment of acute pancreatitis.

**What are the risk factors expected from the study to the participants?**

No risk as this is only an analytical study.

**Whether my participation in the study will be kept confidential ?**

Yes,confidentiality of records will be maintained.

**Is there provision of free treatment for research related injury?**

No intervention will be done for research purpose. So no research related injury is expected.

**Can I withdraw from study when I want?**

Yes. You can withdraw at any time without giving reason and this decicsion will not affect your regular medical care.

**PARTICIPANT CONSENT FORM**

PART 2 OF 2

Participants name: Address:

Title of the project:

**"THE ROLE OF LOW MOLECULAR WEIGHT HEPARIN IN TREATMENT OF ACUTE PANCREATITIS**

The details of the study have been provided to me in writing and explained to me in my own language. I confirm that I have understood the above study and had the opportunity to ask questions. I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without the medical care that will normally be provided by the hospital being affected. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose. I have been given information sheet

giving details of the study. I fully consent to participate in the above study.

I also consent /do not consent to use my stored biological samples for future scientific purposes –if applicable

Signature of the participant: Date:

Signature of the witness: Date

Signature of the investigator: Date:

## KEY TO MASTER CHART

| | | |
|---|---|---|
| **TPR** | **:** | **TEMPERATURE** ( ° C ) |
| **MAP** | **:** | **MEAN ARTERIAL PRESSURE** (mm Hg) |
| **HR** | **:** | **HEART RATE** (ventricular response) |
| **RR** | **:** | **RESPIRATORY RATE** (non ventilated or ventilated) |
| **Na+** | **:** | **SERUM SODIUM** (meg/dl) |
| **K+** | **:** | **SERUM POTASSIUM** (meg/dl) |
| **Creat** | **:** | **SERUM CREATININE** (mg/100 ml) |
| **Hct** | **:** | **HEMATOCRIT** (%) |
| **WBC** | **:** | **WHITE BLOOD COUNT** (total/mm3) (in 1000s) |
| **GCS** | **:** | **GLASGOW COMA SCORE** (GCS) (score 15 minus actual GCS) |

**Total ACUTE PHYSIOLOGY SCORE**(APS)
Sum of the 12 individual variable points

| | | |
|---|---|---|
| CHP | : | Chronic Health Points |

**APACHE SCORE AT DAY 7**

| NAME | ` | TPR (°C) | MAP | HR | RR | NA+ | K+ | CREAT | HCT | WBC | GCS | CHP | Total Score |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| SUBBIAH | 0 | 38.6 | 102 | 86 | 18 | 138 | 3.2(1) | 1.6(1) | 25.8(1) | 7.8 | 0 | 0 | 3 |
| SEKAR | 0 | 38.5(1) | 98 | 96 | 25(1) | 142(1) | 3.5 | 1.2 | 38.7 | 8.6 | 0 | 0 | 4 |
| NAGARAJAN | 0 | 37.6 | 108 | 92 | 28(1) | 152(1) | 3.8 | 1.3 | 26.5(1) | 17.1(1) | 0 | 0 | 4 |
| KAREEM | 0 | 38.8 | 84 | 90 | 20 | 139 | 3.7 | 0.8 | 39.2 | 8.2 | 0 | 0 | 1 |
| PALANISAMY | 1 | 39.6(2) | 113(1) | 96 | 15 | 154(1) | 3.1(1) | 1.8(1) | 26.8(1) | 22.5(2) | 0 | 0 | 10 |
| FAIZAL | 0 | 39(1) | 115(1) | 116(1) | 26(1) | 142 | 3.3(1) | 1.2 | 29.2(1) | 16.8(1) | 1 | 0 | 8 |
| SHANTHAMANI | 2 | 38.5 | 82 | 91 | 18 | 136 | 3.9 | 0.7 | 39.5 | 7.8 | 0 | 0 | 2 |
| KRISHNAN | 1 | 37.2 | 116(1) | 94 | 18 | 142 | 3.2(1) | 1.3 | 28.2(1) | 18.1(1) | 0 | 0 | 4 |
| SHANMUGASUNDARAM | 3 | 37 | 95 | 92 | 22 | 152(1) | 4.1 | 1.6 | 39.1 | 9.2 | 0 | 0 | 5 |
| BALAJI | 0 | 37.6 | 96 | 82 | 16 | 140 | 3.8 | 1.3 | 27.8(2) | 18.4(1) | 0 | 0 | 3 |
| VELUSAMY | 2 | 38.6 | 108(1) | 98 | 22 | 144 | 3.8 | 1.7(1) | 29.1(1) | 19.5(1) | 0 | 0 | 7 |
| GUNASEKARAN | 0 | 38.5(1) | 126(2) | 128(2) | 28(1) | 1328 | 2.8(2) | 0.8 | 30.2(1) | 20.2(1) | 1 | 0 | 11 |
| ARULDAS | 0 | 38.3 | 92 | 86 | 17 | 152(1) | 3.9 | 1.2 | 36.3 | 16.7(1) | 0 | 0 | 2 |
| KARUPPUSAMY | 0 | 38.8(1) | 86 | 117(1) | 27(1) | 142 | 3.8 | 1.1 | 28.7(1) | 24.2(2) | 0 | 0 | 8 |
| SUBBULAKSHMI | 2 | 38.4 | 88 | 118(1) | 26(1) | 139 | 4 | 0.9 | 36.8 | 18.9(1) | 0 | 0 | 5 |
| ALAGIRI | 2 | 36.8 | 80 | 110(2) | 22 | 154(1) | 5 | 2.5(2) | 46.1 | 14.5 | 1 | 0 | 9 |
| MANIKANDAN | 2 | 36 | 110(2) | 120(2) | 30(2) | 120(2) | 5.5(1) | 1.5(2) | 45.9 | 4 | 1 | 0 | 14 |
| JEBAKUMAR | 0 | 37 | 100 | 110(1) | 22 | 150(1) | 3.5 | 1 | 25.5(2) | 14.8 | 0 | 0 | 4 |
| JOSEOH | 0 | 36.8 | 82 | 98 | 18 | 150(1) | 5 | 0.9 | 30.1 | 8 | 0 | 0 | 1 |
| RAVICHANDRAN | 2 | 38.5(1) | 120(2) | 102(2) | 30(1) | 150(1) | 5.5(1) | 1.6(2) | 25(2) | 18.6(1) | 1 | 0 | 16 |
| RADHAKRISHNAN | 2 | 37 | 80 | 90 | 18 | 145 | 3.8 | 1 | 38 | 7 | 1 | 0 | 2 |
| VIDYALAKSHMI | 0 | 37 | 86 | 120(2) | 18 | 136 | 2.4(2) | 1.1 | 26.5(1) | 14.1(1) | 0 | 0 | 7 |
| MANIVEL | 2 | 36.7 | 80 | 120(2) | 18 | 138 | 3.5 | 1 | 32 | 14 | 0 | 0 | 7 |
| PRABHU | 2 | 38 | 110(2) | 100 | 22 | 136 | 3.8 | 2.6(2) | 30.2 | 12 | 0 | 0 | 6 |
| NARASIMMAN | 2 | 36.2 | 80 | 70 | 12 | 155(2) | 2.9(2) | 1.2 | 50 | 15 | 2 | 0 | 10 |
| DHANDAPANI | 2 | 38.4 | 120(2) | 120(2) | 30(1) | 150(1) | 3.4(1) | 1.3 | 46.1 | 20.1(2) | 2 | 0 | 13 |
| MILTON | 0 | 37 | 112(2) | 80 | 22 | 144 | 4.8 | 1.2 | 46(1) | 16.6(1) | 0 | 0 | 5 |
| ESWARAN | 0 | 36.7 | 90 | 78 | 16 | 152(1) | 4.5 | 0.8 | 44.2 | 9 | 1 | 0 | 2 |
| RAFEEQ | 0 | 38.4(1) | 110(2) | 110(2) | 22 | 158(1) | 3.6 | 1.2 | 36 | 12 | 0 | 0 | 6 |
| SELLAYA | 0 | 38 | 88 | 90 | 22 | 145 | 5.6(1) | 1.2 | 40 | 14 | 0 | 0 | 1 |
| JAYARAMAN | 0 | 37.2 | 80 | 82 | 18 | 132 | 4 | 1.2 | 42 | 8 | 1 | 0 | 1 |
| BANUPRIYA | 0 | 37.2 | 90 | 90 | 18 | 153(1) | 3.5 | 1.5(1) | 25(2) | 12 | 0 | 0 | 4 |
| PANDI | 2 | 38 | 100 | 22 | 150 | 3.4(1) | 1.6(2) | 46(1) | 15.1(1) | 2 | 0 | 0 | 9 |

| | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| KALIYAMOORTHY | 2 | 38 | 116(1) | 125(2) | 25(1) | 138 | 4.1 | 1 | 36.5 | 14.2 | 0 | 0 | 6 |
| DINESH | 2 | 38.8(1) | 113(1) | 122(1) | 28(1) | 142 | 3.9 | 1.2 | 20.9(1) | 15.4(1) | 0 | 0 | 9 |
| GANESAN | 0 | 38.1 | 90 | 88 | 18 | 152(1) | 3.8 | 0.8 | 37.2 | 8.2 | 0 | 0 | 1 |
| CHANDRAN | 0 | 38.1(1) | 115(1) | 123(2) | 17 | 140 | 4.2 | 1.3 | 23.2(1) | 7.3 | 1 | 0 | 6 |
| VENKATESH | 1 | 38.5 | 84 | 78 | 20 | 141 | 3.9 | 0.6 | 37.5 | 8.2 | 0 | 0 | 0 |
| KANAGARAJ | 2 | 37.3 | 115(1) | 118(1) | 22 | 141 | 3.9 | 1.2 | 39.2 | 15.5(1) | 1 | 0 | 6 |
| SENNIYAPPAN | 2 | 33.8(2) | 124(2) | 128(2) | 28(1) | 128(1) | 3.2(1) | 1.9(1) | 25.5(1) | 14.8(1) | 1 | 0 | 16 |
| CHINNAKANNU | 0 | 39.2(2) | 92 | 126(2) | 18 | 145 | 4.2 | 1 | 23.2(1) | 12.5 | 0 | 0 | 5 |
| SAMPATH | 0 | 34.2(1) | 90 | 65(1) | 11(1) | 151(1) | 3.9 | 0.8 | 26.5(1) | 18.7(1) | 0 | 0 | 6 |
| VASUMATHI | 0 | 38.4 | 94 | 88 | 20 | 139 | 4.4 | 1.1 | 38.5 | 17.1(1) | 0 | 0 | 1 |
| ARUMUGAM | 0 | 38.8(1) | 116(2) | 112(2) | 28(1) | 154(1) | 3.1(1) | 1.8(1) | 20.6(1) | 18.2(1) | 0 | 0 | 11 |
| PERIYASAMI | 0 | 38.4 | 88 | 112(1) | 18 | 152(1) | 4.3 | 1.3 | 37.6 | 22.2(2) | 0 | 0 | 4 |
| MUNIYAPPAN | 1 | 34(1) | 36(1) | 68(1) | 10(1) | 146 | 4.2 | 1.1 | 24.5(1) | 18.4(1) | 1 | 0 | 9 |
| RAVIKUMAR | 1 | 38.2 | 90 | 84 | 15 | 151(1) | 3.8 | 0.9 | 32.5 | 10.5 | 0 | 0 | 2 |
| PADMANABAN | 0 | 38.4 | 92 | 78 | 16 | 153(1) | 4.1 | 0.7 | 35.9 | 13.5 | 1 | 0 | 2 |
| SHAKILA | 2 | 36 | 92 | 70 | 14 | 138 | 3.8 | 1.2 | 32 | 9 | 0 | 0 | 2 |
| RAJU | 0 | 37.2 | 108 | 90 | 22 | 150(1) | 3.4(1) | 1.5(2) | 35 | 7.2 | 0 | 0 | 4 |
| UDHAYAKUMAR | 1 | 37.4 | 106 | 94 | 25 | 156(1) | 5.6(1) | 1.6(2) | 35 | 13.6 | 0 | 0 | 5 |
| GOWRISHANKAR | 1 | 38 | 100 | 112(2) | 23 | 151 | 4.9 | 1.3 | 40 | 17(1) | 2 | 0 | 6 |
| BALAJI | 2 | 38.6(1) | 113(2) | 105 | 26(1) | 139 | 3.2(1) | 1 | 40 | 15.5 | 0 | 0 | 7 |
| VIJAYARUN | 3 | 38.2 | 116(2) | 115(2) | 29(1) | 145 | 3.6 | 1.1 | 38 | 15.3 | 1 | 0 | 9 |
| GANAPATHY | 0 | 38.2 | 118(2) | 102(2) | 25 | 140 | 3.2(1) | 1.1 | 39 | 21(2) | 0 | 0 | 7 |
| ARULRAJ | 0 | 38 | 110(2) | 98 | 24 | 138 | 3.3(1) | 1 | 37 | 17.1(1) | 0 | 0 | 4 |
| MADHU | 2 | 38 | 112(2) | 115(2) | 30(1) | 119(2) | 3(1) | 1.7(2) | 34 | 18.1(1) | 0 | 0 | 15 |
| JOTHIMANI | 3 | 37.6 | 128(2) | 114(2) | 32(1) | 141 | 3.2(1) | 1 | 27(2) | 18.2(1) | 0 | 0 | 12 |
| NITHYA | 1 | 38.2 | 105 | 106 | 20 | 140 | 5.6 | 1.7(2) | 28.5 | 15.6 | 0 | 0 | 2 |
| PRAKASH | 1 | 38.8(1) | 120(2) | 115(2) | 32(1) | 136(1) | 3.1(1) | 1 | 28.5(2) | 16.5(1) | 1 | 0 | 13 |
| RADHA | 1 | 38.3 | 92 | 94 | 16 | 150(1) | 4.6 | 1 | 40 | 13.5 | 0 | 0 | 2 |
| SARAVANAKUMAR | 1 | 38.6(1) | 113(2) | 124(2) | 11(1) | 150(1) | 5.6(1) | 1 | 36 | 15.2 | 1 | 0 | 10 |
| SARATHA | 0 | 37.4 | 90 | 106 | 11(1) | 140 | 5.7(1) | 1 | 46.5(1) | 17 | 0 | 0 | 4 |
| VELINGIRI | 3 | 38.4(1) | 116(2) | 120(2) | 32(1) | 118(3) | 3.2(1) | 1.9(2) | 40.2 | 1 | 0 | 0 | 18 |
| NATRAJAN | 2 | 38.8(1) | 100 | 96 | 20 | 140 | 3.6 | 1.5(2) | 28(2) | 21(1) | 0 | 0 | 8 |
| SELVI | 1 | 38.2 | 86 | 90 | 16 | 140 | 3.8 | 1 | 38 | 10.6 | 0 | 0 | 0 |
| KALIMUTHU | 1 | 38.5(1) | 64(2) | 67(2) | 11(1) | 140 | 3.2(1) | 1 | 42 | 11 | 0 | 0 | 7 |
| NAGAARAJ | 1 | 38 | 104 | 106 | 26 | 136 | 4 | 1 | 46.1 | 12.2 | 0 | 0 | 2 |
| MANOHARAN | 0 | 38.6(1) | 117(2) | 114(2) | 26(1) | 155(2) | 3.2(1) | 1.7(2) | 37 | 15.2 | 0 | 0 | 11 |
| RAMASAMY | 2 | 37.6 | 90 | 112(2) | 20 | 138 | 2.9(2) | 1.8(2) | 27.8(2) | 21.2(2) | 0 | 0 | 12 |

| | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| SHIVA | 3 | 37.8 | 113(2) | 117(2) | 20 | 148 | 3.9 | 1 | 37 | 11.6 | 0 | 0 | 7 |
| GEETHA | 1 | 38 | 117(2) | 115(2) | 20 | 140 | 3.2(1) | 1.2 | 40.2(1) | 20.1(2) | 0 | 0 | 9 |
| RAMESH | 1 | 38.8(1) | 116(2) | 119(2) | 30(1) | 150(1) | 5.6(1) | 1.9(4) | 19.8(4) | 15.5 | 1 | 0 | 18 |
| SHAHHER AHMED | | | | | death | | | | | | | | |
| VALLI | 0 | 37.6 | 86 | 90 | 18 | 140 | 4 | 1 | 4.2 | 11.6 | 0 | 0 | 0 |
| RAMACHANDRAN | 1 | 37.8 | 110(2) | 112(2) | 26(1) | 140 | 4 | 1.1 | 28.8(1) | 10.5 | 0 | 0 | 7 |
| PARTHIBAN | 0 | 38 | 90 | 116(2) | 28(1) | 129(2) | 2.8(2) | 1.1 | 1 | 17.4(1) | 0 | 0 | 10 |
| MAHALINGAM | 1 | 38 | 112(2) | 113(2) | 26(1) | 140 | 3.4(1) | 1.1 | 28.9(2) | 18.6(1) | 1 | 0 | 11 |
| PRADEEP | 0 | 38.6(1) | 112(2) | 114(2) | 27(1) | 140 | 3.9 | 2.4(3) | 29.2(2) | 17.2(1) | 0 | 0 | 12 |
| GOPI | 1 | 38 | 120(2) | 116(2) | 20 | 128(2) | 3.2(1) | 1.6(2) | 46(1) | 11.6 | 0 | 0 | 11 |
| SHANKAR | 0 | 38.8(1) | 68(2) | 69(2) | 14 | 136 | 5.4 | 1 | 28.8(2) | 11.6 | 0 | 0 | 7 |
| SURESH | 1 | 38.6(1) | 112(2) | 114(2) | 26(1) | 140 | 3.4(2) | 1.1 | 40.1 | 21.1(2) | 0 | 0 | 11 |
| SUNDHAR RAJ | 1 | 37.2 | 122(2) | 112(2) | 25(1) | 152(1) | 5.5(1) | 1.5(2) | 29.1(2) | 15.6 | 0 | 0 | 12 |
| JAMES ARUL RAJ | 1 | 38 | 97 | 102 | 20 | 150(1) | 2.4(4) | 1.2 | 40 | 21.2(2) | 0 | 0 | 9 |
| SASI KUMAR | 0 | 38 | 96 | 100 | 16 | 140 | 4 | 1 | 40.1 | 7.7 | 0 | 0 | 0 |
| JAGADEESH | 2 | 37.6 | 68(2) | 69(2) | 10(1) | 160(3) | 5.7(1) | 1.8(2) | 44.2(2) | 18.6(1) | 0 | 0 | 18 |
| KUMAR | 3 | 37.2 | 98 | 102 | 20 | 128(2) | 3.2(3) | 1 | 27.2 | 15.4 | 0 | 0 | 8 |
| MOHAN | 0 | 37.6 | 99 | 104 | 16 | 150(1) | 3.3(1) | 1.8(2) | 28.8(1) | 15.6 | 0 | 0 | 5 |
| SHAKILA BANU | 1 | 38.8(3) | 117(2) | 112(2) | 25(1) | 129(2) | 3.2(1) | 1.9(2) | 28.6 | 21.1(1) | 0 | 0 | 19 |
| SELVA KUMAR | | | | | | death | | | | | | | |
| KUMARASAMY | 2 | 37.2 | 90 | 90 | 16 | 140 | 4 | 1.1 | 28.8(2) | 15.2 | 0 | 0 | 2 |
| SARAVANAN | 0 | 38.5(1) | 112(2) | 114(2) | 26(1) | 128(1) | 5.7(1) | 3.8(2) | 38.6 | 15.2 | 0 | 0 | 12 |
| STEPHEN RAJ | 0 | 38 | 96 | 102 | 20 | 152(1) | 3.6 | 1.9(2) | 28.7(2) | 15.4 | 0 | 0 | 5 |
| GOVINDHAMMAL | 2 | 38 | 112(2) | 116(2) | 25(1) | 140 | 3.3(1) | 1 | 40.2 | 10.6 | 0 | 0 | 9 |
| NARAYANASAMY | 0 | 37.8 | 90 | 106 | 26(1) | 152(1) | 5.6(1) | 1 | 29(2) | 12.1 | 0 | 0 | 5 |
| MARIMUTHU | 1 | 37.2 | 97 | 90 | 14 | 126(2) | 3.2(1) | 1.2 | 28(2) | 17.2(1) | 0 | 0 | 7 |
| LOGANATHAN | 0 | 37.8 | 100 | 102 | 18 | 136 | 3.2(1) | 1.7(2) | 40 | 13 | 0 | 0 | 3 |
| RAJAN | | | | | | death | | | | | | | |
| SUNDARAM | 1 | 37.6 | 98 | 104 | 22 | 153(1) | 3.1(1) | 1.7(2) | 46.2(2) | 11.6 | 0 | 0 | 6 |
| THIRUMOORTHI | 0 | 37.8 | 102 | 115(2) | 25 | 140 | 2.7(2) | 1.2 | 23.6(2) | 21.2(2) | 0 | 0 | 9 |

**APACHE SCORING AT THE TIME OF ADMISSION**

| NAME | AGE | TPR (°C) | MAP | HR | RR | NA+ | K+ | CREAT | HCT | WBC | GCS | CHP | Total Score |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| SUBBIAH | 0 | 38.7 | 114 (2) | 120 (2) | 28 (1) | 138 | 2.7 (2) | 1.8 (2) | 27.2 (2) | 17.5 (1) | 0 | 0 | 12 |
| SEKAR | 0 | 39. 2(2) | 118 (2) | 124 (2) | 36 (3) | 156 (2) | 3.1 (1) | 1.8 (2) | 25.2 (1) | 14.1 | 0 | 0 | 15 |
| NAGARAJAN | 0 | 37.2 | 112 (2) | 118 (2) | 26 (1) | 160 (1) | 3.9 | 1.5 | 36.6 | 20.1 (2) | 1 | 0 | 9 |
| KAREEM | 0 | 38.2 | 94 | 118 (2) | 20 | 128 (1) | 3.1 (1) | 1.8 (1) | 25.8 (2) | 15 | 0 | 0 | 7 |
| PALANISAMY | 1 | 39.1 (3) | 126 (2) | 130 (2) | 10 (1) | 142 | 5.6 (1) | 2.8 (3) | 29 (2) | 26 (2) | 1 | 0 | 18 |
| FAIZAL | 0 | 39.4 (2) | 128(2) | 138(2) | 38(2) | 154(2) | 5.8(1) | 2.2(2) | 16.2(2) | 25.4(2) | 1 | 0 | 18 |
| SHANTHAMANI | 2 | 38 | 90 | 984 | 16 | 144 | 5.6(1) | 0.7 | 24.8(2) | 18(1) | 0 | 0 | 6 |
| KRISHNAN | 1 | 36.9 | 98 | 126(2) | 24(1) | 124(1) | 2.8(2) | 1.2 | 26 | 16.2(1) | 1 | 0 | 9 |
| SHANMUGASUNDARAM | 3 | 39.2 | 118(2) | 128(2) | 28(1) | 127(2) | 5.1(1) | 3.2(2) | 36.6(2) | 16.2(1) | 1 | 0 | 19 |
| BALAJI | 0 | 37.2 | 96 | 112(1) | 14 | 152(2) | 3.1(1 | 1.7(1) | 26.6(2) | 14.6 | 0 | 0 | 7 |
| VELUSAMY | 2 | 39 (3) | 120(2) | 110(2) | 30(1) | 136 | 3.2(1) | 1.6 | 23.2(2) | 25(2) | 1 | 0 | 16 |
| GUNASEKARAN | 0 | 39.6 (3) | 110(2) | 130(3) | 28(1) | 138 | 2.7(2) | 1.9(2) | 27(2) | 17.5(1) | 1 | 0 | 17 |
| ARULDAS | 0 | 33.2 (2) | 66(2) | 70(2) | 11 | 116(3) | 2.6(3) | 3.2(3) | 24.3(2) | 4 | 1 | 0 | 17 |
| KARUPPUSAMY | 0 | 38.1(1) | 90 | 130(2) | 36(3) | 138 | 3.1(1) | 1.2 | 22.7(2) | 25.4(2) | 1 | 0 | 12 |
| SUBBULAKSHMI | 0 | 37.1 | 110 | 120(1) | 30(1) | 138 | 1.6(2) | 1.2 | 27(2) | 28(2) | 1 | 0 | 10 |
| ALAGIRI | 2 | 36.9 | 94 | 120(2) | 22 | 164(3) | 5 | 2.6(2) | 40.1(1) | 18(1) | 2 | 0 | 13 |
| MANIKANDAN | 2 | 38.2 | 122(2) | 126(2) | 37(2) | 126(2) | 2.4(2) | 2.3(2) | 17(4) | 22.5(2) | 2 | 0 | 22 |
| JEBAKUMAR | 0 | 38 | 123(2) | 130(2) | 26(1) | 157(2) | 3.5 | 1 | 16.5(4) | 16.8(1) | 0 | 0 | 12 |
| JOSEOH | 0 | 37.1 | 84 | 120(2) | 28(1) | 152(1) | 5.5(2) | 0.9 | 26.5(1) | 14.1(1) | 0 | 0 | 8 |
| RAVICHANDRAN | 2 | 39.2(3) | 126(2) | 144(3) | 36(2) | 158(3) | 5.5(1) | 2.7(3) | 18(4) | 20.2(2) | 2 | 0 | 27 |
| RADHAKRISHNAN | 2 | 38.7 | 90 | 98 | 12 | 145 | 3.8 | 0.9 | 36 | 7 | 0 | 0 | 2 |
| VIDYALAKSHMI | 0 | 38 | 90 | 135(2) | 30(2) | 136 | 2.9(3) | 1.1 | 28.9(1) | 17.2(1) | 0 | 0 | 9 |
| MANIVEL | 2 | 36.8 | 96 | 128(2) | 28(1) | 129(2) | 2.7(2) | 0.8 | 26.2(1) | 14.6(1) | 1 | 0 | 12 |
| PRABHU | 2 | 39 (3) | 120(2) | 120(2) | 30(1) | 158(2) | 5.8(1) | 3.2(2) | 20.1(1) | 16.8(1) | 0 | 0 | 17 |
| NARASIMMAN | 2 | 34 (1) | 70 | 68(2) | 9(2) | 165(3) | 2.6(2) | 1.6(1) | 46.2(1) | 18.2(1) | 2 | 0 | 19 |
| DHANDAPANI | 2 | 39.7 (3) | 128(3) | 136(2) | 40(3) | 166(3) | 5.4(1) | 2.0(3) | 48.1(1) | 41.2(4) | 2 | 0 | 27 |
| MILTON | 0 | 39.6 (1) | 119(2) | 98 | 32(1) | 144 | 2.4(4) | 1.6(2) | 18(2) | 18.6(1) | 1 | 0 | 14 |
| ESWARAN | 0 | 34.2(1) | 94 | 68(2) | 14 | 162(3) | 4.2(1) | 0.7 | 46.5(1) | 7 | 2 | 0 | 10 |
| RAFEEQ | 0 | 40 (3) | 124(2) | 144(3) | 39(2) | 162(3) | 2.6(2) | 1.8(2) | 25.5(1) | 18.5(1) | 0 | 0 | 19 |
| SELLAYA | 0 | 38 | 88 | 120(2) | 30(1) | 151(1) | 6.5(2) | 1 | 26.7(1) | 16.3(1) | 0 | 0 | 8 |
| JAYARAMAN | 0 | 38 | 125(2) | 130(2) | 26(1) | 136 | 2.2(2) | 1.2 | 25.8(1) | 9 | 1 | 0 | 9 |
| BANUPRIYA | 0 | 37.5 | 92 | 162(2) | 26(1) | 158(2) | 3.2(1) | 1 | 16.9(2) | 16.6(1) | 0 | 0 | 9 |
| PANDI | 2 | 38.6 (1) | 122 (2) | 128(2) | 38(2) | 126(1) | 2.8(2) | 2.5(2) | 17.0(2) | 24.1(2) | 2 | 0 | 20 |

| | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| KALIYAMOORTHY | 2 | 38.4 | 110(1) | 110(1) | 28(1) | 150(1) | 4.6 | 1 | 26.0(2) | 18.5(1) | 1 | 0 | 10 |
| DINESH | 2 | 39 (3) | 118(2) | 120(2) | 28(1) | 127(2) | 5.8(1) | 3.2(2) | 20.6(2) | 16.8(1) | 1 | 0 | 19 |
| GANESAN | 0 | 38 | 98 | 101(1) | 16 | 152(1) | 3.4(1) | 1.7(1) | 25.6(1) | 17.2(1) | 0 | 0 | 6 |
| CHANDRAN | 0 | 38.1 | 86 | 126(2) | 30(1) | 156(1) | 6.4(3) | 1.1 | 25.9(2) | 10 | 1 | 0 | 11 |
| VENKATESH | 1 | 38 | 90 | 82 | 24(1) | 145 | 3.8 | 1.2 | 36.5 | 16.2(1) | 1 | 0 | 4 |
| KANAGARAJ | 1 | 38.1 | 116(1) | 118(1) | 30(1) | 138 | 3.1(1) | 1.2(2) | 22.8 | 25.2(2) | 1 | 0 | 10 |
| SENNIYAPPAN | 2 | 39.6 (4) | 130(2) | 140(3) | 36(3) | 160(3) | 5.4 | 1.8(1) | 18.4(2) | 22.4(2) | 1 | 0 | 23 |
| CHINNAKANNU | 0 | 36.8 | 94 | 96 | 32(1) | 136 | 1.6(4) | 1.2 | 27.6(2) | 28(2) | 1 | 0 | 10 |
| SAMPATH | 0 | 38 | 88 | 128(2) | 29(1) | 154(1) | 6.5(3) | 0.9 | 26.7(1) | 16 | 1 | 0 | 9 |
| VASUMATHI | 0 | 37 | 98 | 110(1) | 14 | 152(1) | 3.2(1) | 1.7(1) | 24.8(1) | 18.7(1) | 0 | 0 | 6 |
| ARUMUGAM | 0 | 39 (2) | 118(2) | 130(2) | 30(2) | 163(3) | 3.1(1) | 3.2(2) | 16.8(2) | 26.2(3) | 1 | 0 | 19 |
| PERIYASAMI | 0 | 38.6 | 82 | 116(1) | 20 | 154(1) | 4.6 | 1.2 | 26.8(2) | 17(1) | 0 | 0 | 5 |
| MUNIYAPPAN | 1 | 39.2(3) | 120(2) | 120(2) | 30(1) | 130(2) | 2.1(4) | 1.6(2) | 25.8(2) | 16.9(1) | 0 | 0 | 20 |
| RAVIKUMAR | 1 | 38.06 | 110(1) | 88 | 30(1) | 152(1) | 4.2 | 1.2 | 26.8(2) | 11.5 | 0 | 0 | 6 |
| PADMANABAN | 0 | 33 (2) | 65(2) | 68(2) | 12 | 136 | 5.4 | 1.2 | 25.5(2) | 10.8 | 1 | 0 | 9 |
| SHAKILA | 2 | 36 | 90 | 72 | 14 | 138 | 3.1(1) | 1.8(1) | 25.8(1) | 14 | 1 | 0 | 6 |
| RAJU | 0 | 38.6(1) | 120(2) | 120(2) | 25(1) | 152(2) | 2.6(1) | 2.4(2) | 27.2(1) | 15.8(1) | 0 | 0 | 13 |
| UDHAYAKUMAR | 1 | 37 | 108 | 115(2) | 26(1) | 152(1) | 5.8(1) | 1.8(1) | 47(1) | 18(1) | 0 | 0 | 9 |
| GOWRISHANKAR | 1 | 37.4 | 105 | 118(1) | 25(1) | 150(1) | 5 | 1.7(1) | 26(1) | 22.2(2) | 2 | 0 | 10 |
| BALAJI | 2 | 38.6(1) | 116(1) | 114(1) | 28(1) | 136 | 2.8(2) | 1.1 | 24(1) | 16(1) | 0 | 0 | 10 |
| VIJAYARUN | 3 | 38.8(1) | 102 | 126(2) | 32(1) | 158(2) | 3.2(1) | 1 | 21(1) | 13 | 2 | 0 | 13 |
| GANAPATHY | 0 | 37.6 | 92 | 116(1) | 30(1) | 150(1) | 2.6(2) | 1.2 | 25(1) | 24(2) | 1 | 0 | 9 |
| ARULRAJ | 0 | 37.5 | 112(1) | 128(2) | 27(1) | 135 | 3.2(1) | 1.1 | 38 | 12 | 0 | 0 | 5 |
| MADHU | 2 | 38.8 | 115 | 118(1) | 36(2) | 118(2) | 3.2(1) | 1.8(2) | 26(2) | 24(2) | 2 | 0 | 19 |
| JOTHIMANI | 3 | 37.2 | 95 | 116(1) | 36(2) | 140 | 2.6(2) | 0.9 | 28(2) | 26(2) | 3 | 0 | 15 |
| NITHYA | 1 | 37.4 | 108 | 117(1) | 26(1) | 136 | 5.8(1) | 1.8(1) | 24.5(2) | 16.1(1) | 1 | 0 | 9 |
| PRAKASH | 1 | 38.7 (1) | 129(2) | 124(2) | 36(2) | 124(1) | 3.2(1) | 1.9(2) | 28(2) | 16.1(1) | 2 | 0 | 17 |
| RADHA | 1 | 37.6 | 98 | 84 | 16 | 138 | 3.8 | 1.1 | 38 | 18(1) | 2 | 0 | 4 |
| SARAVANAKUMAR | 1 | 33.8 (2) | 114(1) | 68(1) | 10(1) | 159(2) | 5.8(1) | 0.8 | 26(2) | 16(1) | 2 | 0 | 14 |
| SARATHA | 0 | 34.2(1) | 62(1) | 108 | 10(1) | 142 | 5.6(1) | 0.7 | 24.8(2) | 18.5(1) | 0 | 0 | 7 |
| VELINGIRI | 3 | 39.2(3) | 124(2) | 128(2) | 36(3) | 114(3) | 3.0(1) | 1.8(1) | 28(2) | 26.2(2) | 2 | 0 | 24 |
| NATRAJAN | 2 | 34.6 (1) | 90 | 88 | 18 | 124(2) | 6.1(3) | 1.6(1) | 25.6(2) | 28.6(2) | 1 | 0 | 14 |
| SELVI | 1 | 37.6 | 78 | 82 | 14 | 136 | 3.6 | 1.6(1) | 35.6 | 11 | 1 | 0 | 1 |
| KALIMUTHU | 1 | 35.4(1) | 66(2) | 68(2) | 10(1) | 138 | 3.1(1) | 1.2(1) | 24.6(2) | 10.2 | 1 | 0 | 10 |
| NAGAARAJ | 1 | 38.6(1) | 108 | 132(2) | 25(1) | 126(2) | 3.8 | 0.7 | 27.2(2) | 14.8 | 0 | 0 | 8 |
| MANOHARAN | 0 | 38.8(1) | 118(2) | 124(2) | 36(3) | 156(2) | 3.1(1) | 2.5(3) | 25.6(2) | 15.8(1) | 0 | 0 | 15 |
| RAMASAMY | 2 | 37.2 | 88 | 117(2) | 22 | 118(3) | 2.7(2) | 0.8 | 16.5(2) | 26.5(2) | 2 | 0 | 17 |

| | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| SHIVA | 3 | 37.6 | 117(2) | 118(2) | 26(1) | 112(1) | 3.8 | 1.7(1) | 36.6 | 16.4(1) | 0 | 0 | 10 |
| GEETHA | 1 | 38.2 | 114(2) | 128(2) | 28(1) | 138 | 2.6(2) | 2.5(3) | 27.2 | 17.5(1) | 2 | 0 | 13 |
| RAMESH | 1 | 38.6 (1) | 128(2) | 138(2) | 38(3) | 158(2) | 6.6(3) | 1.8(1) | 16.8(4) | 18.2(1) | 2 | 0 | 24 |
| SHAHHER AHMED | 3 | 39.6(2) | 126(2) | 134(2) | 38(2) | 162(3) | 3.1(1) | 3.2(1) | 16.8(2) | 26.2(2) | 5 | 0 | 27 |
| VALLI | 0 | 37.2 | 82 | 88 | 16 | 138 | 3.8 | 1 | 38 | 16.5(1) | 0 | 0 | 1 |
| RAMACHANDRAN | 1 | 38.6 (1) | 112(1) | 118(1) | 28(1) | 152(1) | 4.6 | 1.2 | 26.8(2) | 11.5 | 1 | 0 | 9 |
| PARTHIBAN | 0 | 38.1 | 86 | 132(2) | 36(3) | 128(2) | 2.2(4) | 1.1 | 25.8(2) | 16.9(1) | 0 | 0 | 16 |
| MAHALINGAM | 1 | 38.2 | 116(1) | 118(1) | 36(3) | 138 | 3.1(1) | 0.9 | 22.8(2) | 25.4(2) | 2 | 0 | 14 |
| PRADEEP | 0 | 39.4(3) | 128(2) | 126(2) | 38(3) | 142 | 3.8 | 3.2(3) | 20.8(2) | 16.8(1) | 0 | 0 | 16 |
| GOPI | 1 | 37.6 | 86 | 132(2) | 22 | 118(3) | 2.6(2) | 3.0(3) | 24.3(2) | 16.1(1) | 0 | 0 | 14 |
| SHANKAR | 0 | 33.2 (2) | 65(2) | 68(2) | 10(1) | 132 | 5.5 | 0.9 | 25.5(2) | 10.8 | 0 | 0 | 9 |
| SURESH | 1 | 39.1 (3) | 118(3) | 126(2) | 32(1) | 136 | 3.4(1) | 1.2 | 23.2(2) | 24.5(2) | 1 | 0 | 15 |
| SUNDHAR RAJ | 1 | 36.9 | 115(2) | 126(2) | 29(1) | 158(2) | 6.5(2) | 1.6(1) | 28.6(2) | 18.6(1) | 2 | 0 | 17 |
| JAMES ARUL RAJ | 1 | 37.1 | 119(1) | 116(1) | 32(1) | 138 | 1.6(4) | 1.2 | 28(2) | 28.0(2) | 2 | 0 | 14 |
| SASI KUMAR | 0 | 38.7(1) | 94 | 98 | 14 | 144 | 3.8 | 0.7 | 38 | 7 | 0 | 0 | 1 |
| JAGADEESH | 2 | 34.2(1) | 68(1) | 68(1) | 8(2) | 162(3) | 5.8(1) | 2.5(3) | 46.5(1) | 26.5(2) | 3 | 0 | 23 |
| KUMAR | 3 | 36.9 | 94 | 128(2) | 26(1) | 124(1) | 2.6(2) | 0.8 | 26.2(1) | 16.7(1) | 1 | 0 | 12 |
| MOHAN | 0 | 37.2 | 98 | 116(1) | 14 | 152(1) | 3.2(1) | 1.7(1) | 25.8(1) | 17.2(1) | 0 | 0 | 6 |
| SHAKILA BANU | 1 | 38.7(1) | 124(2) | 128(2) | 39(2) | 128(1) | 2.6(2) | 2.5(3) | 17.1(4) | 24.5(2) | 3 | 0 | 24 |
| SELVA KUMAR | 3 | 39.2(3( | 126(2) | 144(3) | 38(2) | 162(3) | 5.6(1) | 1.8(1) | 18(2) | 22.2(2) | 5 | 0 | 30 |
| KUMARASAMY | 2 | 36.9 | 96 | 128(2) | 14 | 156(2) | 3.6 | 3.2(1) | 20.8(2) | 18.5(1) | 0 | 0 | 5 |
| SARAVANAN | 0 | 39.9(3) | 118(2) | 84 | 28(1) | 127(2) | 5.8(1) | 1 | 20.6(2) | 16.8(1) | 0 | 0 | 17 |
| STEPHEN RAJ | 0 | 37.5 | 92 | 117(1) | 26(1) | 158(2) | 3.6 | 1.2 | 16.9(4) | 16.8(1) | 0 | 0 | 9 |
| GOVINDHAMMAL | 2 | 38.2 | 125(2) | 132(2) | 28(1) | 136 | 2.8(2) | 1.1 | 25.9(2) | 10 | 2 | 0 | 13 |
| NARAYANASAMY | 0 | 38 | 86 | 129(2) | 30(1) | 158 | 6.5(3) | 0.9 | 26.7(1) | 16.1(1) | 0 | 0 | 10 |
| MARIMUTHU | 1 | 35.2(1) | 88 | 64(1) | 12 | 132 | 2.8(2) | 1.3 | 27.6(2) | 18.9(1) | 2 | 0 | 10 |
| LOGANATHAN | 0 | 38 | 98 | 116(1) | 29(1) | 128(1) | 3.8(1) | 1.8(1) | 25.8(2) | 14.2 | 0 | 0 | 7 |
| RAJAN | 3 | 39.4(2) | 126(2) | 138(2) | 36(2) | 156(2) | 5.8(1) | 2.1(2) | 16.2(2) | 25.2(2) | 5 | 0 | 12 |
| SUNDARAM | 1 | 38.3 | 112(2) | 136(1) | 33(1) | 152(1) | 3.2(1) | 1.6(1) | 28.2(2) | 10.5 | 2 | 0 | 26 |
| THIRUMOORTHI | 0 | 38 | 94 | 113(2) | 26(1) | 136 | 2.6(2) | 1.9(4) | 20.9(2) | 24.5(2) | 1 | 0 | 13 |

| NAME | AGE | AGE CODE | SEX | DOA | DOD | DAYS IN HOSPITAL | APACHE ON AD | APACHE ON DAY 7 | OUTCOME | RECOVERY CODE | HEPARIN | HEP CODE |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| SUBBIAH | 29 | 1 | MALE | 3.6.16 | 10.6.16 | 7 | 12 | 8 | RECOVERED | 1 | YES | 1 |
| SEKAR | 32 | 2 | MALE | 3.6.16 | 10.6.16 | 7 | 15 | 4 | RECOVERED | 1 | YES | 1 |
| NAGARAJAN | 32 | 2 | MALE | 10.6.16 | 18.6.16 | 8 | 9 | 3 | RECOVERED | 1 | YES | 1 |
| KAREEM | 42 | 3 | MALE | 10.6.16 | 17.6.16 | 7 | 7 | 0 | RECOVERED | 1 | YES | 1 |
| PALANISAMY | 40 | 2 | MALE | 1.7.16 | 12.7.16 | 11 | 17 | 9 | RECOVERED | 1 | YES | 1 |
| FAIZAL | 43 | 3 | MALE | 8.7.16 | 20.7.16 | 12 | 18 | 8 | RECOVERED | 1 | YES | 1 |
| SHANTHAMANI | 46 | 3 | FEMALE | 22.7.16 | 29.7.16 | 7 | 6 | 2 | RECOVERED | 1 | YES | 1 |
| KRISHNAN | 41 | 3 | MALE | 29.7.16 | 6.8.16 | 7 | 9 | 4 | RECOVERED | 1 | YES | 1 |
| SHANMUGASUNDARAM | 57 | 4 | MALE | 5.8.16 | 14.8.16 | 9 | 19 | 5 | RECOVERED | 1 | YES | 1 |
| BALAJI | 29 | 1 | MALE | 12.8.16 | 19.8.16 | 7 | 7 | 2 | RECOVERED | 1 | YES | 1 |
| VELUSAMY | 50 | 3 | MALE | 26.8.16 | 8.9.16 | 13 | 16 | 7 | RECOVERED | 1 | YES | 1 |
| GUNASEKARAN | 33 | 2 | MALE | 2.9.16 | 14.9.16 | 12 | 11 | 11 | RECOVERED | 1 | YES | 1 |
| ARULDAS | 38 | 2 | MALE | 9.9.16 | 19.9.16 | 10 | 17 | 2 | RECOVERED | 1 | YES | 1 |
| KARUPPUSAMY | 53 | 4 | MALE | 23.9.16 | 30.9.16 | 7 | 12 | 6 | RECOVERED | 1 | YES | 1 |
| SUBBULAKSHMI | 43 | 3 | FEMALE | 30.9.16 | 7.10.16 | 7 | 10 | 3 | RECOVERED | 1 | YES | 1 |
| ALAGIRI | 53 | 4 | MALE | 27.1.17 | 6.2.17 | 10 | 13 | 9 | RECOVERED | 1 | YES | 1 |
| MANIKANDAN | 46 | 3 | MALE | 3.2.17 | 18.2.17 | 15 | 22 | 4 | RECOVERED | 1 | YES | 1 |
| JEBAKUMAR | 28 | 1 | MALE | 10.2.17 | 21.2.17 | 11 | 12 | 4 | RECOVERED | 1 | YES | 1 |
| JOSEOH | 30 | 1 | MALE | 17.2.17 | 25.2.17 | 7 | 8 | 1 | RECOVERED | 1 | YES | 1 |
| RAVICHANDRAN | 50 | 3 | MALE | 24.2.17 | 13.3.17 | 17 | 27 | 16 | RECOVERED | 1 | YES | 1 |
| RADHAKRISHNAN | 53 | 4 | MALE | 3.3.17 | 10.3.17 | 7 | 5 | 2 | RECOVERED | 1 | YES | 1 |
| VIDYALAKSHMI | 26 | 1 | FEMALE | 17.3.17 | 24.3.17 | 7 | 9 | 7 | RECOVERED | 1 | YES | 1 |
| MANIVEL | 52 | 4 | MALE | 24.3.17 | 1.4.17 | 8 | 12 | 5 | RECOVERED | 1 | YES | 1 |
| PRABHU | 42 | 3 | MALE | 7.4.17 | 17.4.17 | 10 | 17 | 6 | RECOVERED | 1 | YES | 1 |
| NARASIMMAN | 50 | 3 | MALE | 14.4.17 | 27.4.17 | 13 | 19 | 10 | RECOVERED | 1 | YES | 1 |
| DHANDAPANI | 51 | 4 | MALE | 21.4.17 | 5.5.17 | 14 | 27 | 13 | RECOVERED | 1 | YES | 1 |
| MILTON | 35 | 2 | MALE | 28.4.17 | 8.5.17 | 10 | 14 | 5 | RECOVERED | 1 | YES | 1 |
| ESWARAN | 38 | 2 | MALE | 5.5.17 | 13.5.17 | 8 | 10 | 2 | RECOVERED | 1 | YES | 1 |
| RAFEEQ | 19 | 1 | MALE | 12.5.17 | 21.5.17 | 9 | 19 | 6 | RECOVERED | 1 | YES | 1 |
| SELLAYA | 43 | 3 | MALE | 26.5.17 | 2.6.17 | 7 | 8 | 1 | RECOVERED | 1 | YES | 1 |
| JAYARAMAN | 45 | 3 | MALE | 2.6.17 | 9.6.17 | 7 | 9 | 1 | RECOVERED | 1 | YES | 1 |
| BANUPRIYA | 40 | 2 | FEMALE | 9.6.17 | 16.6.17 | 7 | 9 | 4 | RECOVERED | 1 | YES | 1 |
| PANDI | 52 | 4 | MALE | 23.6.17 | 3.7.17 | 10 | 20 | 9 | RECOVERED | 1 | YES | 1 |
| KALIYAMOORTHY | 52 | 4 | MALE | 7.10.16 | 16.10.16 | 9 | 10 | 6 | RECOVERED | 1 | YES | 1 |
| DINESH | 51 | 4 | MALE | 14.10.16 | 22.10.16 | 8 | 19 | 9 | RECOVERED | 1 | YES | 1 |
| GANESAN | 28 | 1 | MALE | 28.10.16 | 4.11.16 | 7 | 6 | 1 | RECOVERED | 1 | YES | 1 |
| CHANDRAN | 30 | 1 | MALE | 4.11.16 | 12.11.16 | 8 | 10 | 6 | RECOVERED | 1 | YES | 1 |

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| VENKATESH | 36 | 2 | MALE | 18.11.16 | 25.11.16 | 7 | 4 | 0 | RECOVERED | 1 | YES | 1 |
| KANAGARAJ | 40 | 2 | MALE | 25.11.16 | 2.12.16 | 7 | 10 | 6 | RECOVERED | 1 | YES | 1 |
| SENNIYAPPAN | 48 | 3 | MALE | 2.12.16 | 16.12.16 | 14 | 23 | 15 | RECOVERED | 1 | YES | 1 |
| CHINNAKANNU | 24 | 1 | MALE | 2.12.16 | 12.12.16 | 10 | 10 | 5 | RECOVERED | 1 | YES | 1 |
| SAMPATH | 22 | 1 | MALE | 9.12.16 | 17.12.16 | 8 | 9 | 6 | RECOVERED | 1 | YES | 1 |
| VASUMATHI | 39 | 2 | FEMALE | 16.12.16 | 23.12.16 | 7 | 6 | 1 | RECOVERED | 1 | YES | 1 |
| ARUMUGAM | 41 | 3 | MALE | 23.12.16 | 7.1.17 | 15 | 19 | 11 | RECOVERED | 1 | YES | 1 |
| PERIYASAMI | 29 | 1 | MALE | 30.12.16 | 6.1.17 | 7 | 5 | 4 | RECOVERED | 1 | YES | 1 |
| MUNIYAPPAN | 34 | 2 | MALE | 30.12.16 | 11.1.17 | 12 | 19 | 3 | RECOVERED | 1 | YES | 1 |
| RAVIKUMAR | 36 | 2 | MALE | 6.1.17 | 13.1.17 | 7 | 5 | 1 | RECOVERED | 1 | YES | 1 |
| PADMANABAN | 39 | 2 | MALE | 6.1.17 | 13.1.17 | 7 | 9 | 2 | RECOVERED | 1 | YES | 1 |
| SHAKILA | 47 | 3 | FEMALE | 13.1.17 | 20.1.17 | 7 | 6 | 2 | RECOVERED | 1 | YES | 1 |
| RAJU | 43 | 3 | MALE | 20.1.17 | 29.1.17 | 9 | 13 | 4 | RECOVERED | 1 | YES | 1 |
| UDHAYAKUMAR | 30 | 1 | MALE | 1.6.16 | 9.6.16 | 8 | 9 | 5 | RECOVERED | 1 | NO | 2 |
| GOWRISHANKAR | 27 | 1 | MALE | 2.6.16 | 14.6.16 | 12 | 10 | 6 | RECOVERED | 1 | NO | 2 |
| BALAJI | 38 | 2 | MALE | 8.6.16 | 21.6.16 | 13 | 10 | 7 | RECOVERED | 1 | NO | 2 |
| VIJAYARUN | 59 | 4 | MALE | 16.6.16 | 1.7.16 | 15 | 13 | 9 | RECOVERED | 1 | NO | 2 |
| GANAPATHY | 38 | 2 | MALE | 28.6.16 | 7.7.16 | 9 | 9 | 7 | RECOVERED | 1 | NO | 2 |
| ARULRAJ | 40 | 2 | MALE | 1.7.16 | 9.7.16 | 8 | 5 | 4 | RECOVERED | 1 | NO | 2 |
| MADHU | 48 | 3 | MALE | 10.7.16 | 31.7.16 | 21 | 1 | 15 | RECOVERED | 1 | NO | 2 |
| JOTHIMANI | 56 | 4 | MALE | 24.7.16 | 13.8.16 | 25 | 15 | 12 | RECOVERED | 1 | NO | 2 |
| NITHYA | 24 | 1 | FEMALE | 3.8.16 | 13.8.16 | 10 | 9 | 5 | RECOVERED | 1 | NO | 2 |
| PRAKASH | 20 | 1 | MALE | 10.8.16 | 25.8.16 | 15 | 17 | 13 | RECOVERED | 1 | NO | 2 |
| RADHA | 42 | 3 | FEMALE | 20.8.6 | 27.8.16 | 7 | 4 | 2 | RECOVERED | 1 | NO | 2 |
| SARAVANAKUMAR | 36 | 2 | MALE | 30.8.16 | 9.9.16 | 10 | 14 | 10 | RECOVERED | 1 | NO | 2 |
| SARATHA | 41 | 3 | FEMALE | 4.9.16 | 12.9.16 | 8 | 7 | 4 | RECOVERED | 1 | NO | 2 |
| VELINGIRI | 60 | 4 | MALE | 10.9.16 | | 2 | 24 | | DIED | 2 | NO | 2 |
| NATRAJAN | 46 | 3 | MALE | 20.9.16 | 29.9.16 | 9 | 14 | 8 | RECOVERED | 1 | NO | 2 |
| SELVI | 40 | 2 | FEMALE | 14.3.17 | 21.3.17 | 7 | 1 | 0 | RECOVERED | 1 | NO | 2 |
| KALIMUTHU | 40 | 2 | MALE | 16.3.17 | 25.3.17 | 9 | 10 | 7 | RECOVERED | 1 | NO | 2 |
| NAGAARAJ | 43 | 3 | MALE | 6.4.17 | 16.4.17 | 10 | 8 | 2 | RECOVERED | 1 | NO | 2 |
| MANOHARAN | 22 | 1 | MALE | 10.4.17 | 25.4.17 | 15 | 15 | 11 | RECOVERED | 1 | NO | 2 |
| RAMASAMY | 49 | 3 | MALE | 14.4.17 | 2.5.17 | 18 | 17 | 12 | RECOVERED | 1 | NO | 2 |
| SHIVA | 58 | 4 | MALE | 29.4.17 | 13.5.17 | 14 | 10 | 7 | RECOVERED | 1 | NO | 2 |
| GEETHA | 36 | 2 | FEMALE | 5.5.17 | 17.5.17 | 17 | 13 | 9 | RECOVERED | 1 | NO | 2 |
| RAMESH | 44 | 3 | MALE | 10.5.17 | 30.5.17 | 20 | 24 | 18 | RECOVERED | 1 | NO | 2 |
| SHAHHER AHMED | 62 | 4 | MALE | 20.5.17 | | 2 | 27 | | DIED | 2 | NO | 2 |
| VALLI | 40 | 2 | FEMALE | 24.5.17 | 31.5.17 | 7 | 1 | 0 | RECOVERED | 1 | NO | 2 |
| RAMACHANDRAN | 17 | 1 | MALE | 2.6.17 | 10.6.17 | 8 | 9 | 7 | RECOVERED | 1 | NO | 2 |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|
| PARTHIBAN | 30 | 1 | MALE | 6.6.17 | 22.6.17 | 16 | 16 | 10 | RECOVERED | 1 | NO | 2 |
| MAHALINGAM | 39 | 2 | MALE | 10.6.17 | 25.6.17 | 15 | 14 | 11 | RECOVERED | 1 | NO | 2 |
| PRADEEP | 40 | 2 | MALE | 14.6.17 | 29.6.17 | 15 | 16 | 12 | RECOVERED | 1 | NO | 2 |
| GOPI | 43 | 3 | MALE | 20.6.17 | 3.7.17 | 13 | 14 | 11 | RECOVERED | 1 | NO | 2 |
| SHANKAR | 25 | 1 | MALE | 24.6.17 | 4.7.17 | 10 | 9 | 7 | RECOVERED | 1 | NO | 2 |
| SURESH | 28 | 1 | MALE | 26.6.17 | 12.6.17 | 16 | 15 | 11 | RECOVERED | 1 | NO | 2 |
| SUNDHAR RAJ | 27 | 1 | MALE | 25.9.16 | 9.10.16 | 15 | 17 | 12 | RECOVERED | 1 | NO | 2 |
| JAMES ARUL RAJ | 30 | 1 | MALE | 1.10.16 | 17.10.16 | 16 | 14 | 9 | RECOVERED | 1 | NO | 2 |
| SASI KUMAR | 20 | 1 | MALE | 10.10.16 | 17.10.16 | 7 | 1 | 0 | RECOVERED | 1 | NO | 2 |
| JAGADEESH | 46 | 3 | MALE | 2.11.16 | 24.11.16 | 22 | 23 | 18 | RECOVERED | 1 | NO | 2 |
| KUMAR | 59 | 4 | MALE | 10.11.16 | 19.11.16 | 9 | 12 | 8 | RECOVERED | 1 | NO | 2 |
| MOHAN | 34 | 2 | MALE | 20.11.16 | 28.11.16 | 8 | 6 | 5 | RECOVERED | 1 | NO | 2 |
| SHAKILA BANU | 47 | 3 | FEMALE | 27.11.16 | 17.12.16 | 20 | 24 | 19 | RECOVERED | 1 | NO | 2 |
| SELVA KUMAR | 63 | 4 | MALE | 1.12.16 | | 2 | 30 | | DIED | 2 | NO | 2 |
| KUMARASAMY | 33 | 2 | MALE | 5.12.16 | 12.12.16 | 7 | 5 | 2 | RECOVERED | 1 | NO | 2 |
| SARAVANAN | 50 | 3 | MALE | 20.12.16 | 9.1.17 | 20 | 17 | 12 | RECOVERED | 1 | NO | 2 |
| STEPHEN RAJ | 38 | 2 | MALE | 1.1.17 | 11.1.17 | 10 | 9 | 5 | RECOVERED | 1 | NO | 2 |
| GOVINDHAMMAL | 49 | 3 | FEMALE | 13.1.17 | 22.1.17 | 9 | 13 | 9 | RECOVERED | 1 | NO | 2 |
| NARAYANASAMY | 40 | 2 | MALE | 22.1.17 | 30.1.17 | 8 | 10 | 5 | RECOVERED | 1 | NO | 2 |
| MARIMUTHU | 42 | 3 | MALE | 1.2.17 | 9.2.17 | 8 | 10 | 2 | RECOVERED | 1 | NO | 2 |
| LOGANATHAN | 28 | 1 | MALE | 5.2.17 | 12.2.17 | 7 | 7 | 3 | RECOVERED | 1 | NO | 2 |
| RAJAN | 63 | 4 | MALE | 15.2.17 | | 2 | 26 | | DIED | 2 | NO | 2 |
| SUNDARAM | 34 | 2 | MALE | 21.2.17 | 2.3.17 | 9 | 12 | 6 | RECOVERED | 1 | NO | 2 |
| THIRUMOORTHI | 38 | 2 | MALE | 10.3.17 | 20.3.17 | 10 | 13 | 9 | RECOVERED | 1 | NO | 2 |